The Journal of the American Association of Nurse Anesthetists

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Volume XXII AUGUST 1954 Number Three

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The new Puritan flush-mounted

WALL OUTLET ASSEMBLIES

for central supply lines are superior to all older types because:

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Other combinations available for both concealed and exposed piping

Safety **Non-interchangeable valves connect only to proper equipment** . . . Apparatus quick-connects with straight thrust (no twisting) and is mechanically locked into position . . . Releases instantly at touch of push button, and exclusive secondary locking device "catches" equipment if handled carelessly . . . Automatic port cover protects outlet when idle and discourages tampering.

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The Journal of the American Association of Nurse Anesthetists is published quarterly by the American Association of Nurse Anesthetists, 116 S. Michigan Ave., Chicago 3, Ill. Entered as second class matter, May 14, 1945, at the post office at Chicago, Ill., under the act of March 3, 1879. Address all communications relative to editorial and advertising matters to the Editor, 116 S. Michigan Ave., Chicago 3, Ill. Subscription prices: to members, 50c a year; to nonmembers and institutions $1.50 a year; single copies, 50c. The opinions expressed in the columns of the Journal are those of the contributors and are not to be construed as reflecting the views of the American Association of Nurse Anesthetists.

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The Journal OF THE
American Association of Nurse Anesthetists
Vol. XXII No. 3 August, 1954

What Does A.A.N.A. Do For Me?

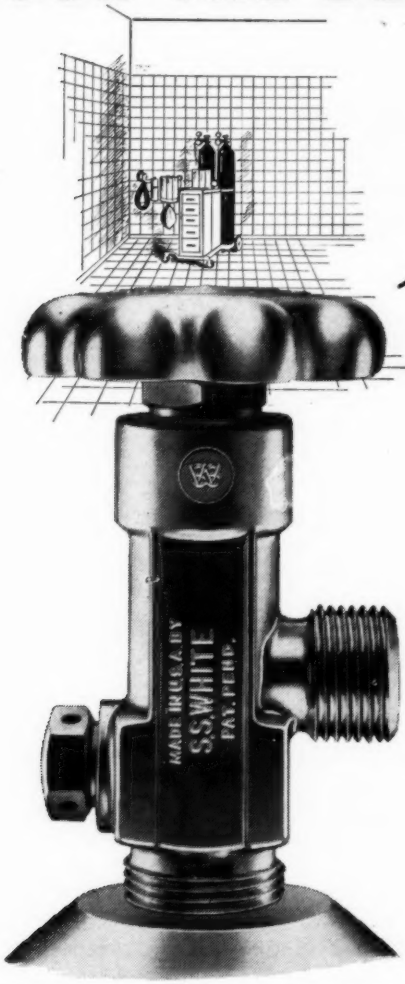
Asking of this question is the favorite method for "baiting" the members who work for A.A.N.A. by those who do not. Of tangible things that A.A.N.A. does for a member there are not many: a Journal that brings to each member scientific information on modern trends in anesthesia; a News Bulletin that brings information on association affairs; a membership card that in time past has been mainly for identification for voting privileges but which is becoming more and more important as more hospital Administrators demand that Nurse Anesthetists must be members of A.A.N.A.

Intangible benefits of membership far outweigh the tangible. Some of the intangibles become apparent when reading our publications, reading the History of A.A.N.A. and by studying committee reports. To fully appreciate the intangibles we must consider "I" not as an individual but as one of many, banded together for the betterment of all, both now and in the future. In the betterment of all there is inevitable betterment for the individual.

A better question would be to ask "What do I do for A.A.N.A.?" If the only answer is "I pay my dues" it is time to re-evaluate the problem. Money is essential and dues have financed all of our projects. But someone has done more than pay dues or we would not have attained our present professional standing. Any organization functions by the pooling of the thoughts and ideas of its members. If some members fail to contribute either thoughts or ideas or fail to study and comprehend the thoughts and ideas of others, they are missing the opportunity to know what A.A.N.A. does for its members. Among us are many members capable of assisting in the work and projects of the organization and who would probably like to do so.

Have you made it known by word or deed that you are willing to contribute to the association's projects? No one needs to sit on the side lines! Be **interested** and **active** in your State association. There you have an opportunity to prove your abilities and interests. There you begin to participate in national affairs. Suggestions for committee appointments can stem from your interest at state meetings. Have you shown an interest in the nominations and elections? Have you contributed an item for Notes and Case Reports in the J.A.A.N.A.? This section of our Journal was originated for the members' participa-

USE THE BEST



The valve illustrated is for oxygen therapy. Flush type valves for smaller cylinders are also available.

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Embodying the most advanced and up-to-date features of modern valve engineering, S.S.White Easy Operating *valves are designed to meet the exacting standards of gas anesthesiology and therapy. They fulfill every requirement of safety, convenience and dependability.*

Ask your gas supplier to equip your cylinders with these outstanding medical valves. They're tested and approved for anesthetic, therapeutic and resuscitating gas service.

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tion but often goes begging for material. An item on a pertinent subject for Opinion Review would give a true cross section of opinion. A paper prepared and presented at a local or state meeting might easily find a place in the Journal. Any of these activities would help A.A.N.A. find you. It is unreasonably selfish for more than 6,000 to depend year after year, on the same people to give of their time and effort chiefly because those who appoint committees don't know who and where the capable members are. Make yourselves known! Do something for A.A.N.A. Only in this way can you appreciate what A.A.N.A. does for you,—**Minnie V. Haas, R.N.**

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in the Respiratory and Circulatory Emergencies of Intravenous Barbiturate Anesthesia.

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Twenty-First Annual Convention American Association of Nurse Anesthetists

September 13-16, 1954

CHICAGO

Hotel Headquarters — Sheraton Hotel

Monday, Wednesday and Thursday sessions will be held at North Hall 2, Navy Pier. Sunday sessions will be held in the Tally-ho Room of the Sheraton Hotel. Tuesday sessions will be held in the Ballroom of the Sheraton Hotel.

PROGRAM

Sunday, September 12

8:00 A.M.-5:00 P.M.—**Registration**
A.A.N.A. Registration—Outside Tally-ho Room, Sheraton Hotel

9:30 A.M.-5:30 P.M.—**Registration†**
A.H.A. Registration—A.H.A. Headquarters, Palmer House

9:00 A.M.— **Assembly of Directors of Schools of Anesthesia***
Tally-ho Room—Sheraton Hotel
Clarene A. Carmichael, R.N., B.S.
Educational Director, A.A.N.A.
Presiding Officer

Evaluation of Tests
Arthur Assum, Ph.D.
Director, Department of Psychometry
Roosevelt College, Chicago, Ill.

2:00 P.M.— **Assembly of Directors of Schools of Anesthesia***
Helen Lamb Powell, R.N.
Chairman, Education Committee
Presiding Officer

Teaching Can be Fun!
Clarene A. Carmichael, R.N., B.S.
and
Cameron W. Meredith, Ph.D.
Professor of Education
Northwestern University, Evanston, Ill.

---

†A.H.A. Registration also Saturday, September 11

*Although this program is of specific interest to Directors of Schools of Anesthesia, ALL members are invited to attend these sessions.

A new and versatile local anesthetic agent

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HYDROCHLORIDE (HEXYLCAINE HYDROCHLORIDE)

CYCLAINE has now been under clinical investigation for more than five years. Its three primary qualities, of interest to anesthesiologists, are these:

Rapid onset of action

Longer duration than procaine

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CYCLAINE is useful for the following types of anesthesia:

Topical · Endoscopic procedures
Infiltration
Nerve block · Caudal
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Investigators[2-10] agree on these qualities of CYCLAINE:

Effective in low concentration

Rapid in onset

Provides prolonged anesthesia

Wide margin of safety

Of further interest to the anesthesiologist is the fact that CYCLAINE has proved *not* to be antagonistic to para-aminosalicylic acid or streptomycin.[11]

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Injection of CYCLAINE Hydrochloride Hexylcaine Hydrochloride 1% supplied in 30 cc. vials, containing 10 mg. CYCLAINE Hydrochloride per cc. (No. 3118). (Isotonic Solution).

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For spinal anesthesia:
Injection of CYCLAINE Hydrochloride Hexylcaine Hydrochloride 2.5% with 10% Dextrose is supplied in 2 cc. ampuls, containing 25 mg. of CYCLAINE Hydrochloride per cc. with 10% Dextrose (No. 3119). (Hyperbaric Solution).

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REFERENCES:
1. Report of the Council on Pharmacy and Chemistry of the A.M.A.: In press. 2. Hudon, F., and Jacques, A.: Proc. Canad. Anesthetists Soc. (1952) p. 34. 3. Orkin, L. R., and Rovenstine, E. A.: Anesthesiology 13:465 (Sept.) 1952. 4. Crawford, O. B.: Anesthesiology 14:278 (May) 1953. 5. Lee, L. W., Davis, E., Jr., and Barmore, J.: To be published. 6. Arcuri, R. A., Newman, W., and Burstein, C. L.: Anesthesiology 14:46 (Jan.) 1953. 7. Hudon, F., and Gendron, P.: Laval med. 18:145 (Feb.) 1953. 8. Ray, E. S., and Vinson, P. P.: Anesthesiology 14:315 (May) 1953. 9. Clark, R. E., Orkin, L. R., and Rovenstine, E. A.: To be published. 10. Stark, E. J.: To be published. 11. Proctor, A. M., et al.: Paper presented at 11th Conference on Chemotherapy of Tuberculosis, V. A., St. Louis, 1952.

Monday, September 13

8:00 A.M.— **Registration**

A.H.A. Registration—Registration Desk, Navy Pier

A.A.N.A. Registration—A.A.N.A. Exhibit Booth 228, Navy Pier

9:00 A.M.— **Assembly of Directors of Schools of Anesthesia***

North Hall 2, Navy Pier

Clarene A. Carmichael, R.N., B.S.
Presiding Officer

Discussion of Class Outlines for Schools of Anesthesia

2:00 P.M. **General Session**

North Hall 2, Navy Pier

Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Invocation

Catherine Gallagher, R.N.
Chicago, Ill.

Address of Welcome

Josephine B. Bunch, R.N.
President, A.A.N.A.

Address of Welcome from A.H.A.

Charles U. Letourneau. M.D.
Secretary
Council on Professional Practice

2:15 P.M.— Exire O'Day, R.N.
President, Illinois Association of Nurse Anesthetists
Presiding Officer

What Every Anesthetist Should Know About the Heart

Samuel Rosenthal. M.D.
Assistant Professor of Medicine
Marquette University School of Medicine
Milwaukee, Wis.

Cardiac Surgery in Relation to Anesthesia

Edward E. Avery, M.D.
Associate in Surgery
Northwestern University
Chicago, Illinois

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*Although this program is of specific interest to Directors of Schools of Anesthesia, ALL members are invited to attend these sessions.

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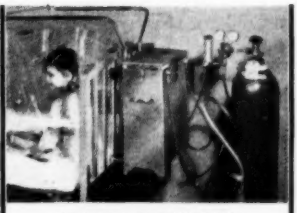

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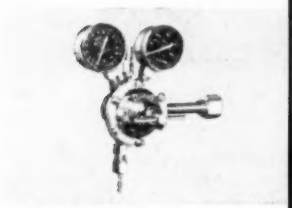
NCG's Plymouth* Tent is widely preferred for children and infants because of its therapeutic merit plus its ease of operation, flexibility of use and low cost. Compact, lightweight, ideal for cubicles. Effectively used as a croup tent.

Because of the key role inhalation therapy increasingly plays in hospital procedures, men responsible for the selection of inhalation therapy equipment weigh carefully the considerations that govern its design and manufacture.

NCG Medical Division works in close cooperation with eminent medical authorities in the design of its inhalation therapy equipment. This equipment has resulted in new effectiveness of attack on respiratory diseases of various types. Hospital and clinic directors not only have reported such successes but have voiced sincere approval of NCG equipment. These facts can be laid before you in greater detail by your NCG representative.



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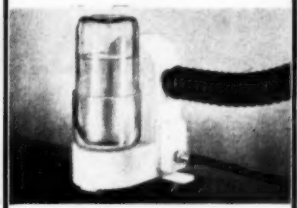
NCG's new "6400" oxygen regulator represents an important advance in regulator design. It delivers the unvarying rate of flow characteristic of the finest 2-stage regulators, yet is comparable in cost to single-stage types.



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The NCG Microfier* is the only continuously operating nebulizer that delivers oxygen super-saturated with moisture particles of microscopic size. Has a remarkable record of successes in respiratory diseases, post operative therapy, etc.

*Trade Mark

NEW OPEN-TOP OXYGEN TENT

Combines with NCG "Microfier" (described at left) to provide high concentrations of super-saturated oxygen. Open top facilitates nursing care. Inexpensive. May be used with or without antibiotic and detergent solutions.

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One of these easily portable units in the Emergency Room and on each floor provides valuable protection against emergencies. Unit consists of truck for small (Type E) cylinder, regulator, mask and tubing. Moderately priced.

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Monday, September 13

7:00 P.M.— **State Night Dinner**
Ballroom, Sheraton Hotel
Marie McLaughlin, R.N.
Chairman, Convention Committee
Presiding Officer

Tuesday, September 14

9:00 A.M.— **Business Session**
Ballroom, Sheraton Hotel
Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Call to Order
Report of Approval of Minutes Committee
Reports of Officers
Reports of Standing Committees

11:00 A.M. to 1:00 P.M.— **Election of Officers**

2:00 P.M.— **Business Session**
Ballroom, Sheraton Hotel
Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Reports of Standing Committees
Reports of Special Committees
Unfinished Business
New Business

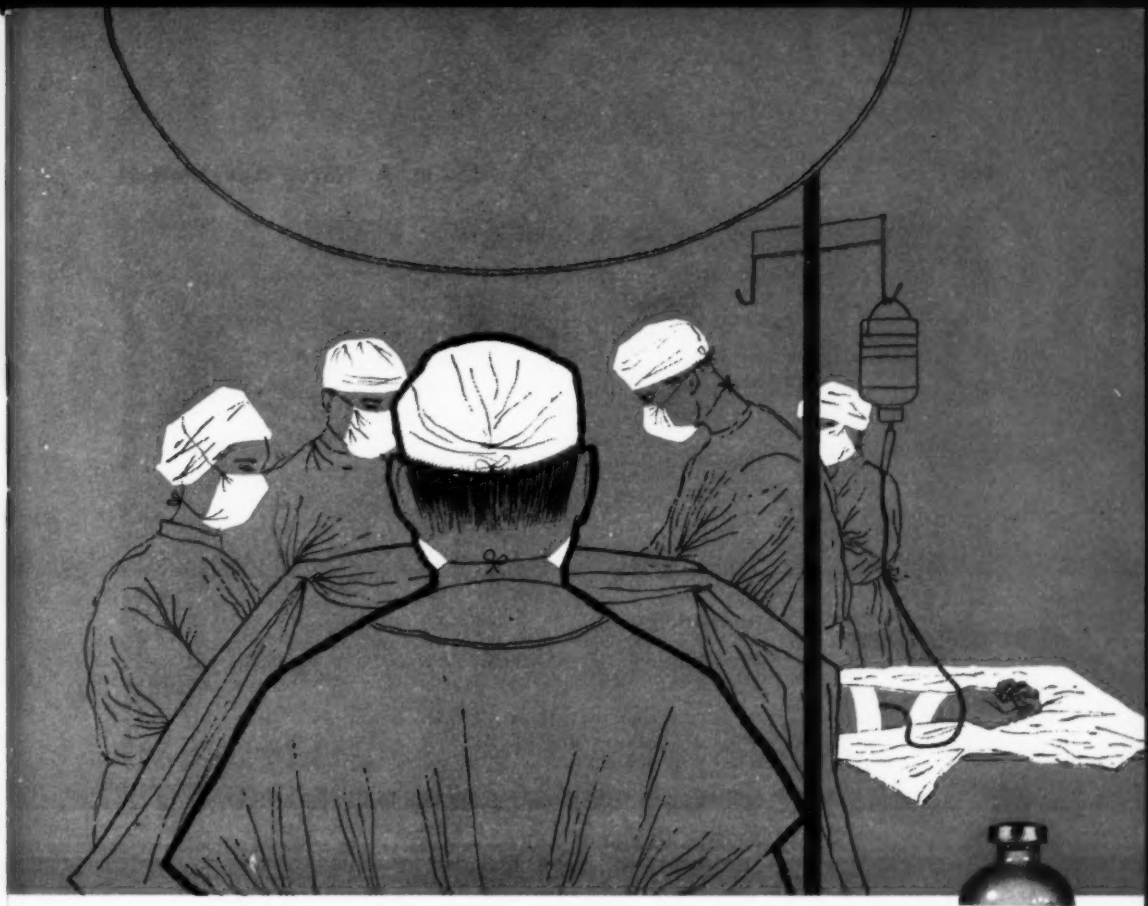
Wednesday, September 15

No sessions have been planned for Wednesday morning in order that the members may have the opportunity to visit the Exhibits, sight-see, visit the Executive Office, or do whatever else they may please.

2:00 P.M.— **General Session**
North Hall 2, Navy Pier
Phyllis Conley, R.N.
President, West Virginia Association of
Nurse Anesthetists
Presiding Officer

Anesthesia for the Negro Patient
Mary F. Poe, M.D.
Director, Department of Anesthesiology
John Gaston Hospital, University of Tennessee
Memphis, Tenn.

Anesthesia for the Paraplegic Patient
R. Anthony Tella, M.D.
Chief, Anesthesia Service
Veterans Administration Hospital
Wood, Wis.



for "...smooth, rapid induction..."[1]
"...a quiet state of sleep..."[2]
"...emergence...quiet, pleasant..."[3]

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ultrashort-acting intravenous anesthetic

SURITAL sodium (thiamylal sodium, Parke-Davis) is noted for producing smooth anesthesia with little excitement during induction or recovery. Laryngospasm is infrequent and there is relative freedom from bronchospasm and respiratory or circulatory depression.

Detailed information on SURITAL sodium is available on request.

1. Helrich, M.; Papper, E. M., & Rovenstine, E. A.: *Anesthesiology* 11:33, 1950. 2. Stephen, C. R., & Martin, R.: *North Carolina M. J.* 12:501, 1951. 3. Phillips, H. S.: *Anesth. & Analg.* 32:56, 1953.

MEDICAMENTA VERA

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Wednesday, September 15 (Cont.)

Anesthesia and Airway Problems in Patients with Poliomyelitis

Ernst Trier Morch, M.D., Ph.D., D.A.
Professor and Director of Anesthesia
University of Chicago
Chicago, Ill.

7:30 P.M. **Banquet**

Ballroom, Sheraton Hotel

Josephine B. Bunch, R.N.
President, A.A.N.A.
Presiding Officer

Invocation

Presentation of A.A.N.A. Award of Appreciation to the

Hospital Sisters of the Third Order of St. Francis,
Sr. Rudolpha, R.N.,
Director, School of Anesthesia
St. John's Hospital, Springfield, Ill., receiving

Presentation of Pin to

Josephine B. Bunch, R.N.,
Shriner's Hospital
Portland, Ore., outgoing president

Thursday, September 16

9:00 A.M.— **General Session**

North Hall 2, Navy Pier

Sylvia E. Abraham, R.N.
President, Nebraska Association of
Nurse Anesthetists
Presiding Officer

Induced Hypotension

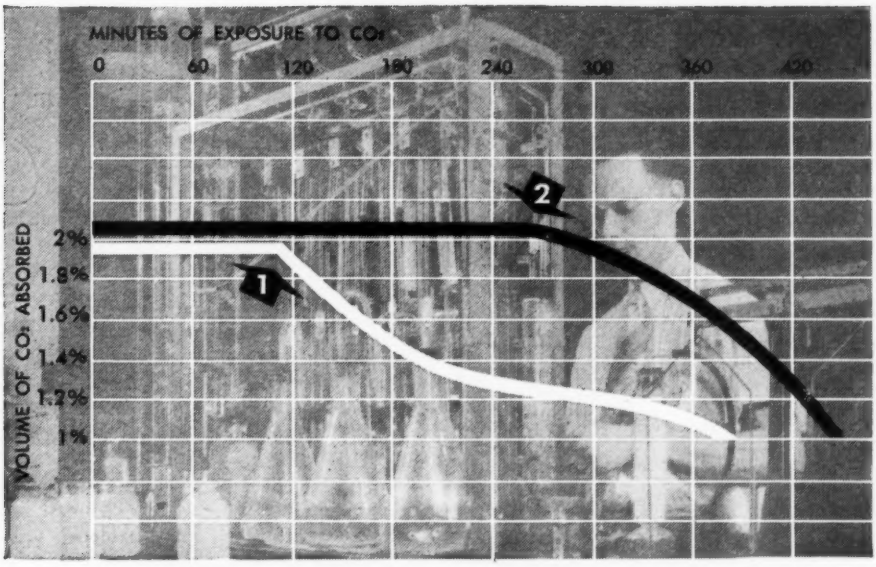
Donald E. Hale, M.D.
Chief, Department of Anesthesia
Cleveland Clinic
Cleveland, Ohio

An Age of Analgesia

John S. Lundy, M.D.
Senior Consultant, Section of Anesthesiology
Mayo Clinic
Rochester, Minn.

Question and Answer Period

John S. Lundy, M.D.
Donald E. Hale, M.D.



1. **BREAK POINT of COMMERCIALLY AVAILABLE BARIUM HYDROXIDE TYPE of GAS ABSORBENT**
After 115 minutes of exposure to CO_2, unabsorbed traces begin to pass through the barium hydroxide type gas absorbent.

2. **BREAK POINT of SODASORB**
Not until 140 minutes later do unabsorbed traces of CO_2 begin to pass through SODASORB. A total of 255 minutes of complete CO_2 absorption.

EVIDENCE OF SODASORB'S LONGER LIFE

The charted results shown above were obtained from quantitative tests designed to give scientifically absolute measurements.

Equal volumes of a barium hydroxide type of CO_2 absorbent and SODASORB (Wilson Soda Lime) were placed in canisters 15 centimeters deep, then reacted with an air stream simulating the exhalation of a patient. The air mixture, containing 2% by volume of CO_2 and moisture equivalent to 85% humidity at 20°C., was passed through the canisters at 3.52 litres per minute until the first trace of unabsorbed CO_2 appeared in detection apparatus.

PLEASE NOTE: Samples tested, though equal in volume, were not equal in weight. The porous SODASORB granules weighed 189 grams; the denser barium hydroxide-type pellets weighed 228. In other words, 228 grams of barium hydroxide-type gas absorbent had a break point of 115 minutes, while the break point of only 189 grams of SODASORB was more than 2 *times as long!* Incidentally, samples of every batch of SODASORB are subjected to this same test as standard control procedure.

Order SODASORB from your hospital supply house, or write for free technical data.

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Thursday, September 16

2:00 P.M.— **General Session**

North Hall 2, Navy Pier
Rita Loran, R.N.
President, Montana Association of Nurse Anesthetists
Presiding Officer

The Short-Acting Muscle Relaxants

Robert B. Sweet, M.D.
Associate Professor of Anesthesiology
University of Michigan School of Medicine
Ann Arbor, Mich.

Anesthesia for Geriatric Surgery

Ethelyn McFarland, R.N.
Elaine McMurdie, R.N.
Anesthesia Department
Butterworth Hospital
Grand Rapids, Mich.

4:00 P.M.— **Unfinished Business**

4:15 P.M.— **Adjournment of General Session**
Council Session*

Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

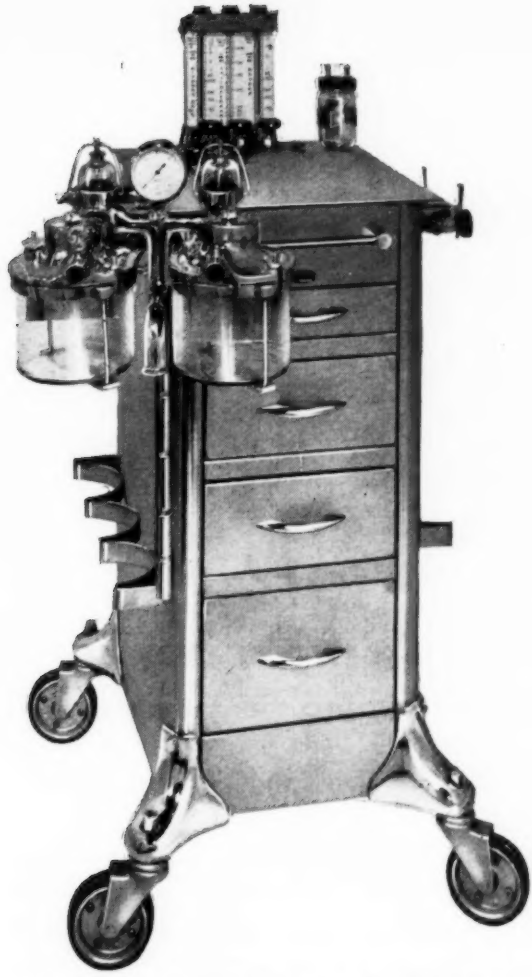
*Although the Council, as provided by the By-Laws of the A.A.N.A., consists of officers and standing committees of national and state associations, the Council session is open to all members of the A.A.N.A., and will begin immediately after the adjournment of the General Session.

Call to the Convention

As provided for in the Bylaws of this Association, and at the direction of Mrs. Josephine B. Bunch, president, we hereby issue this official call to the members for the annual meeting to be held in Chicago, September 13-16, 1954, at the Navy Pier. The annual business session will be held on Tuesday, September 14, in the Ballroom of the Sheraton Hotel.

Accomplished at the Executive Offices, 116 South Michigan Avenue, Chicago 3, Illinois, this second day of July, 1954.

(Signed) FLORENCE A. MCQUILLEN, R.N.
Executive Director



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The Model 460 Series is mounted in a beautiful four drawer Stainless Steel Cabinet, with large ball bearing casters. Embodying INDIVIDUALLY CALIBRATED FLOWMETERS, as well as Tank Pressure Gauges, Reducing Regulators, Yoke Shut-Offs, Lung Pressure Gauge, Built-in Aspirator, as well as an efficient CO_2 Absorber and Ether Vaporizer.

Price of Three Gas Unit as shown **$735.00 FOB Chicago, Ill.**

COMPLETE STOCK OF ENDOTRACHEAL ACCESSORIES

PORTEX TUBES **INFLATABLE CUFFS**

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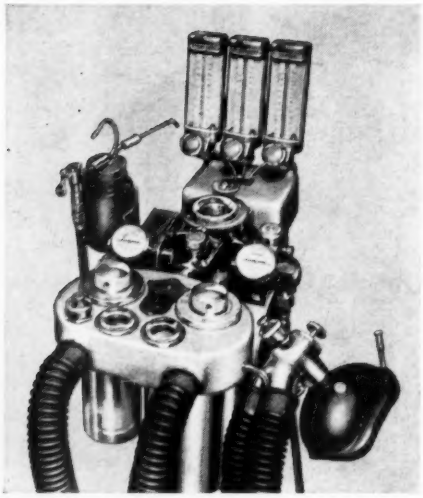

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Also, this combination provides the intermittent automatic flow which is *the most economical method* yet devised for fractional rebreathing techniques.

It's the ultimate in modern convenience and economy.

McKesson

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The Abuse of Parenteral Fluids During Operation and in the Postoperative Period

Robert E. L. Berry, M.D.*
Ann Arbor, Michigan

Parenteral fluid administration to surgical patients under anesthesia and during the postoperative period when they are unable to eat and drink has achieved general acceptance as the best available method of attempting to maintain body homeostasis during these periods of oral deprivation and stress. Such fluid repletion and maintenance has undergone considerable improvement since Latta first described the dramatic improvement of cholera patients to parenterally administered sodium chloride and sodium bicarbonate solutions in 1831.[1] A vast amount of experimental and clinical investigation has importantly added to our knowledge of the pathologic physiology of the loss of body fluids. Much, however, remains to be learned. Because of defects in our knowledge many phases of this subject should be approached with considered awareness of our ignorance.

It is desirable to periodically consider resultant deleterious effects of mistakes in the use of parenteral fluids. Extensive use inevitably is followed by abuse in a minor percent of cases. For the most part such abuses are controllable but certain unphysiological responses are undoubtedly related to factors of which our knowledge is incomplete.

It cannot be overemphasized that considerable variability may exist in the response of patients to the same parenteral fluid. The nature of the disease or injury, the qualitative and quantitive factors of fluid loss and the nutritional status are preoperative considerations importantly related to the response to supporting fluids administered during and after operation. Anoxia, hypotension, magnitude of operation, blood loss and the duration and type of anesthesia are importantly related not only to response after operation but, indeed, during the operation itself. Postoperatively, gastro-intestinal dysfunction, pulmonary and cardiovascular complications, infection, progressive malnutrition, improper fluid replacement and gen-

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Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 4, 1954.

	Heart Sounds	Peripheral Pulse	Respirations	Venous Filling	Color and Warmth of Extremities
Cardiac Arrest	Absent	Absent	Rate and amplitude depressed; may cease.	Diminished or absent.	Cold and cyanotic; may become livid or mottled.
Temporary Anesthetization of Vasomotor Center (e.g. Pentothal)	Present; may be decreased.	Decreased volume; normal or increased rate.	Apnea or markedly depressed.	Usually normal, may be increased.	Variable; may be no change.
Unsuspected contracted blood volume.	Present; may be decreased.	Marked diminution in volume; may be absent; increased rate.	Unchanged or may be depressed.	Marked diminution	Cold and cyanotic
Temporary anesthetization of autonomic nerves (e.g. spinal)	Present	Unchanged or rate may slow.	No significant change.	Good; may be increased.	Warm and of good color.
Pituitary or Adrenal Insufficiency	Present	Variable; usually decrease in volume and increased rate.	No significant change.	Variable; usually decreased.	Variable; usually cold but may be warm and of good color.

Table 1. Causes of Sudden Blood Pressure Fall during Induction of Anesthesia and Differential Diagnosis.

eral debility may profoundly modify the response to parenteral fluids.

When all these variables are considered, it becomes obvious that there is no place for rigid dogma, standard rules or standing orders in the use of parenteral fluids. Each patient must be evaluated as an individual problem.

Use and Misuse of Fluids During Operation

The anesthetist may be faced by a sudden drop in blood pressure during induction with a general or regional anesthetic. There are five major causes of this phenomenon: 1) cardiac arrest; 2) excessive concentration of an anesthetic with temporary paralysis of central nervous system centers controlling vasoconstriction (e.g. too rapid administration of sodium pentothal); 3) contracted blood volume previously unrecognized; 4) regional pooling of blood probably secondary to anesthetization of autonomic nerves; and 5) disturbances in homeostasis secondary to pituitary or adrenal insufficiency. (Table 1)

Cardiac arrest is most dramatic and little difficulty should be encountered in making the diagnosis as long as continued awareness of this serious condition is maintained. Blood pressure and peripheral pulses are unobtainable. Heart sounds are absent, respirations may be barely perceptible or cease and there is onset of rapid cyanosis. Serious or irreversible effects upon the central nervous system result if circulation is not reestablished in 4-5 minutes. By long odds, the most important part of treatment is immediate cardiac massage. The chest is rapidly entered, the pericardium opened and manual massage at 110-120 per minute instituted. This is adjunctively supported by intratracheal oxygen administered by artificial respiration using the anesthetic bag. Whole blood transfusion, if available, is immediately started. Adrenalin should **NOT** be given until the status of the myocardium has been observed although small doses in procaine solution may have to be given directly into the myocardium if no one is immediately available to enter the chest and begin cardiac massage. Subsequent myocardial injection of adrenalin, procaine, calcium chloride and use of the defibrillator may be necessary under certain circumstances to restore cardiac beat and rhythm.

Temporary paralysis of central nervous system centers controlling vasoconstriction, as a general rule, rapidly responds to cessation of anesthetic administration and artificial respiration provided that a lethal amount has not been inadvertently administered.

A contracted blood volume, previously unrecognized, may manifest itself by a sudden drop in pressure, a rapid rise in pulse rate and intense peripheral vasoconstriction. This has been described as "cold shock" because of vaso-constriction with cooling of the extremities. The anesthetic should be immediately abandoned, oxygen administered, and whole blood transfusion started. Careful observation is essential until the blood pressure and pulse have become stabilized. Unless the operation is an extreme emer-

gency it should be abandoned, the patient returned to his room and subsequent blood volume studies carried out and necessary transfusions administered before operation is attempted again. Even if blood volumes determinations show that they are "normal", blood transfusions should be given because despite such "normal" blood volumes there is usually attendant deficit of red blood cell mass and plasma proteins.[2]

Pooling of blood in certain areas of the body, probably in response to anesthetization of segments of the autonomic nervous system, is characterized by a fall in blood pressure but the extremities remain warm and the veins full. The pulse does not significantly change and rate may actually decrease. This type of "warm shock" is usually of little significance and will respond adequately to small doses of vasoconstrictor drugs. Some observers have felt, however, that it is unnecessary to bring up blood pressure by this means because the overall effect during long and extensive procedures may prove more deleterious than beneficial.[3] In substantiation of this observation we have only to observe that marked hypotension can be deliberately induced during operation by hypotensive drugs without necessarily producing injurious effects.

Relative pituitary and/or adrenal insufficiency may be precipitated by stress induced by anesthesia and/or operation.[4] It is an infrequent occurrence, however. The clinical picture is variable but tends to resemble "cold shock" although warm extremities have been observed. Failure to respond to vasoconstrictor drugs and blood transfusions together with high blood eosinophil counts are characteristic of the condition. Dramatic response to whole adrenal cortical extract in large doses confirms the diagnosis. The operation should be abandoned and proper preparation with ACTH and cortisone carried out before operation is again attempted.

It is essential that a large gauge needle (#18 or larger) be firmly secured in a vein, preferably of the forearm or antecubital fossa, at the beginning of an operation. Because of the ever present potential for need of blood during operation the best practical method of starting such an infusion is by the intravenous route. In infants a cutdown is usually necessary in order to insert a needle of desired size.

A carbohydrate (glucose, fructose or a combination of both) and water or a sodium salt solution is usually used to initiate intravenous infusion. The basic function of such procedures is to provide a readily available means of giving medication and supporting fluids, particularly blood, when they become necessary. Blood may be safely added to the infusion without washing out the tubing with saline if the infusion has been started with glucose and water solution. Although it has been pointed out that certain unpleasant side effects may follow the mixing of blood and glucose and water solutions,[5] such has proven to be a universally safe practice at this clinic.

The continuance of carbohydrate and water or sodium salt solutions as the only supportive

measure during a long and difficult operation is a mistake and may lead to serious sequelae. Carbohydrate and water solutions provide for insensible loss during operation (evaporation from the skin and water vapor from the lungs) and certain amounts of glucose and/or fructose to exercise a protein sparing effect and aid in combating ketosis. They do not defend extracellular fluid volume, however, and therefore cannot provide effective help for support of circulation. (Table 2) The maximum amount of this solution that can be given safely during an operation has wide range of variability. It is much safer to administer water if subsequent need develops rather than to be faced postoperatively with a situation of water overloading induced during operation. To be on the safe side is to keep the patient on the dry side. Before more than 1000 cc. are administered careful consideration should be given as to the need because we cannot accurately predict how a given patient will tolerate excesses of water administered during anesthesia.

In contradistinction to carbohydrate and water solutions, sodium salt solutions possess the ability to effectively aid in the defence of extracellular fluid (blood plasma and interstitial fluid found between blood vessels and body cells). But because these solutions contain no colloid substance, they too are relatively ineffective in supporting circulation during long and difficult operations. Most of the standard solutions of this type contain about 9 grams of sodium salts per 1000 cc. Unless there have been pre-operative signs of extracellular fluid deficit or a history of loss by vomiting, diarrhea or intestinal fistulae only limited amounts of sodium salts should be given a patient under anesthesia. It is much better to change to carbohydrate and water solutions than to indiscriminately give 9-18 grams of sodium salts under these circumstances. (Table 2) It would not be unreasonable to assume that the variables induced by anesthesia and operation would make the fate of such an infusion unpredictable.[6;7;8] Much of this sodium will have to be excreted as the need for it may be minimal. Furthermore, certain patients are unable to tolerate sodium salts well in the postoperative period and such an infusion may well represent the initiating point for serious fluid balance disturbances.[9] The average well nourished, well hydrated patient, coming to operation, has little need of this load of salt as an added burden on kidney and circulation during both the operation and postoperative period. Unless there is physiological justification it is best not to use saline solutions to initiate intravenous infusion as the salt is little needed, may be poorly tolerated and if the duration of operation exceeds pre-operative expectations another liter of solution may well be infused with the good intention of keeping the needle open. The administration of 18 grams of salt under such circumstances may well represent 2-3 times the ordinary daily intake of salt in the food and many times more than the actual need. Little justification can be offered for imposing such a burden as

Solution	Advantages	Disadvantages
Carbohydrate and water	1. Provides water for insensible loss and urine formation. 2. Aids in prevention of ketosis and has protein sparing effect. 3. Unnecessary sodium cannot be administered.	1. Does not support circulation or extracellular fluid. 2. Excessive administration produces unnecessary water loading which the postoperative kidney may not well tolerate.
Isotonic sodium salt	1. If previous sodium salt deficit has existed such solutions will aid in replacement. 2. Will support extracellular fluid volume.	1. Does not efficiently support circulation. 2. The administration of only 1000 cc. provides 9 grams of sodium salts which may be inefficiently excreted in the postoperative period and result in unnecessary expansion of the extracellular fluid volume postoperatively. 3. Water for insensible loss and urine formation is inefficiently provided.
Whole Blood	1. Most efficient replacement for blood lost during operation. 2. Best method of combating most types of shock.	1. Increased cost. 2. Hazards inherent to whole blood transfusions (mismatch, etc.) 3. Potential dangers from over-transfusion.
Plasma and plasma substitutes	1. Cheap and easily available. 2. Can be used to cover small blood losses.	1. Do not support circulation as efficiently as whole blood. 2. Do not replace red blood cells lost during operation. 3. Homologous serum reactions still too common.

Table 2. Advantage and Disadvantage of Parenteral Fluids Commonly Used During Operation.

the postoperative response of body fluid homeostasis, cardiopulmonary and renal systems remains unpredictable.[6;7]

In prolonged and difficult operations there is only one type of infusion that fulfills approximately ideal requirements and that is whole blood. There is no substitute for blood because that is essentially what is being lost during operation. Whole blood, furthermore, has the highest efficiency for supporting the circulation during any type of stress.[3]

Although it has been pointed out that there are certain abuses of blood transfusion,[10] this technic has actually achieved remarkable safety. Blood should be given as it is being lost. There is ample evidence to show that transfusions, withheld during operation, and given postoperatively as the need becomes evident, have lessened efficiency.[11] There is little justification for withholding blood transfusions particularly if the anticipated blood loss is greater than 500 cc. If blood is not readily available from an organized blood bank, then plasma, dextran or polyvinylpyrrolidone may offer temporary adequate support of plasma volume during a reasonably small blood loss.[12;13] But operations of considerable magnitude should not be attempted unless correctly cross-matched whole blood is available.

Blood replacement during operation should at least parallel and even slightly exceed the estimated loss. This is best done by carefully weighing sponges and packs, accurate estimation of loss in suction bottles and reasonable estimation of blood loss on drapes and towels.[14] It is well recognized that an empirical estimate without gravimetric measurement tends to grossly underestimate actual blood loss. Increasing pulse rate, unstable or falling blood pressure, peripheral vasoconstriction, weak pulsations of major vessels, color and temperature changes in the extremities, falling venous pressure and visual evidence of improper oxygenation may suggest that blood replacement has been insufficient. The need for further transfusion is obvious.

Although under experimental conditions young healthy adults may tolerate as much as 3000 cc. of whole blood by rapid infusion without deleterious effects[3] the patient under anesthesia and undergoing operation does not fit into this category. Inadequate blood replacement during operation is a well recognized entity. It is not so generally appreciated, however, that excessive amounts of blood may be given during operation even though the "apparent" need exists. This is particularly prone to occur during long operations requiring numerous transfusions in order to replace blood loss. Such over-transfusion may be very dangerous as apparently adjustments which permit the circulation of normal unanesthetized individuals to tolerate over-transfusion have lessened efficiency in anesthetized patients undergoing operation.

Blood pressure, pulse, type, rate and amplitude of respiration have long been considered guides of paramount importance in determining the patient's general condition during operation. Careful observation and maintenance

of accepted suitable criteria as regards cardio - respiratory systems have guided a great many patients through long and difficult operations. Routine brachial artery blood pressures, however, measure only the obvious and that is brachial artery pressure. It does not tell the whole story about certain important factors that may begin to undergo unsuspected changes as the anesthetic and operation become prolonged. Cardiac output, general vascular resistance, the status of constriction or dilatation of major venous systems and excessive adrenomedullary response (adrenalin) are considerations that are not necessarily reflected in the pulse and routine recordings of brachial artery pressure.

Many aspects of circulatory hemodynamics during anesthesia remain poorly understood but it becomes progressively more evident, under certain circumstances, that complete reliance upon brachial artery pressure as the index of adequacy of circulation is not foolproof. During long and difficult operations it has been repeatedly observed that blood transfusions become progressively less effective in maintaining or stabilizing blood pressure. Under cyclopropane anesthesia about 70% of patients will develop generalized vaso-constriction if the operation is prolonged.[15] The significance of this phenomenon is not well understood but it could possibly represent excessive adrenomedullary response. The net result is that brachial artery pressure may fall and transfusions given to stabilize blood pressure can actually be in excess of measured blood loss. During operation the following may be suggestive of slow development of this syndrome: 1) after 3-4 hours operating time blood pressure and pulse become unstable despite apparent adquate blood replacement; 2) adequate surgical anesthesia cannot be maintained without producing unstable blood pressure; 3) narrowing of the pulse pressure with relative increase in the diastolic phase; 4) drops in blood pressure that require larger amounts of blood than should be reasonably anticipated to bring the pressure back to "normal"; 5) increased respiratory effort and obvious lack of proper oxygenation even when breathing almost pure oxygen; and 6) distention of veins in the operating field and "venous bleeding"; observation of major arteries reveal them to be constricted. (Table 3) Subacute heart failure, congestive atelectasis and pulmonary edema have been terms used to describe various phases of this condition. Immediately postoperatively there may be intense cyanosis and marked dyspnea. Peripheral veins may or may not be distended. The paradox of high venous and low arterial pressures, under certain circumstances, may be treated by intra-arterial transfusions. Phlebotomy may be of help if peripheral veins are distended. It is of little help, however, if they are not. It is much better to prevent this condition than attempt to treat it after it occurs. Cyanosis, dyspnea, tachypnea, low arterial and venous pressures is a particularly malignant syndrome. Positive pressure oxygen administered by the BLB mask may be all that can be done. The mortality of

Table 3. Signs of Seriously Disturbed Circulatory Homeostasis That May Result in Overtransfusion Because of the "Apparent" Need For Blood Based Upon Brachial Artery Pressure.

1. After 3-5 hours of operating time the brachial artery pressure and peripheral pulse become unstable despite apparent adequate blood replacement.
2. In spite of adequate blood replacement anesthesia cannot be maintained at suitable depth without producing a fall in brachial artery pressure.
3. Progressively larger amounts of blood are needed to bring brachial artery blood pressure back to "normal".
4. Narrowing of the pulse pressure with relative increase in diastolic phases.
5. Increasing respiratory effort and obvious lack of proper oxygenation of the blood even when breathing pure oxygen.
6. Distention of veins in the operating field and "venous bleeding" despite observation that major arteries are constricted.
7. Postoperative hypotension with associated dyspnea, cyanosis and tachypnea without venous distention or physical and X-ray signs of pulmonary congestion.

congestive atelectasis is high and unless dyspnea and cyanosis can be relieved within a few hours most of these patients will die.

The Postoperative Period

Fluid balance considerations related to operation are not necessarily at an end following recovery of consciousness and stabilization of pulse and blood pressure. Untoward events occurring during operation may have seriously compromised the ability of the postoperative patient to perform the basic functions of respiration, circulation and excretion. Because of the necessity for early evaluation of the effects of operation fluid administration for the first twenty-four hours should be as bland as possible. At the University of Michigan Hospital fluids administered during the day of operation generally are limited to replacement of the blood loss in addition to 1000 - 1500 cc of 5% glucose in distilled water to cover insensible loss and provide water for urine formation.

During the postoperative period repeated careful clinical evaluation and objective interpretation of certain laboratory examinations of the blood and urine are the best available methods for evaluating fluid balance status. Only in this manner can the number of errors in fluid management be kept to a minimum particularly if the period of deprivation of food and oral fluids is prolonged. All effects should be directed towards getting the patient eating and drinking normally. Any other method is a poor substitute and should be considered as such.

Water

Water is needed to provide for urine formation and insensible loss through the lungs and skin in addition to water lost extrarenally by vomiting, diarrhea, intubation and fistulous drainage from the gastro-intestinal tract. Insensible water loss (water vapor of lungs and evaporation from the skin) is obligatory. The urine volume and specific gravity, in the majority of cases, can be depended upon to reflect the water needs of the patient.

It is not unusual to observe a temporary oliguria during the first 24-30 hours postoperatively particularly following operations of considerable magnitude.[9] "Ideal" urine outputs of 1000-1500 cc. during the first twenty-four hours do not necessarily have to be attained. Indeed an attempt to provide this volume may actually be deleterious, as temporary depression of renal function may not permit the kidneys to excrete large loads of water.

The subsequent water needs administered as 5-10% glucose should not be subjected to standard rules as there can be tremendous variability in water excreted during the postoperative period. In the absence of excessive sweating 1500-2500 cc of water is usually sufficient for essential needs. Urine outputs in excess of 700 cc per 24 hours are usually adequate for excretion of the nitrogenous metabolic wastes.

Persistent oliguria during the postoperative period is always a very serious complication. It is routine at our hospital to carefully check the urine specific gravity of all 24 hour volumes less than 500 cc. This should be done **Before** fluids for that day are ordered. The finding of a low urine specific gravity should be a warning signal that possible serious renal damage has occurred. Urine specific gravity of 1.010 or lower with continued oliguria usually signifies serious kidney damage. Important causes of such injury are anoxia, hypotension and incompatible blood transfusion. It is useless and extremely hazardous to "force" fluids under these circumstances because the kidneys will not excrete urine. The infused fluid accumulates in the body and may produce such serious complications as pulmonary edema with associated failure of the circulation and cerebral edema (confusion, convulsions, disorientation etc.). Fluid intake should be sharply limited to less than the anticipated insensible loss until spontaneous recovery of kidney function occurs which is usually initiated by a sharp increase in urine output despite continued limitation of intake. Dialysis on an artificial kidney may eventually be necessary.

Urine specific gravities of 1.020 or greater suggest that an adequate amount of water has not been given and additional water such as 5-10% glucose should be administered that day.

Adequate urine outputs in the postoperative period are only adequate when considered in the light of the fluid intake. Glucose and water solutions intravenously tend to produce water dehydration in certain patients. Because of this it is extremely important that oral intake of

food and drink be resumed as soon as possible.

Salt

The routine use of glucose in "normal" saline as the only supportive fluid in the postoperative period is a pernicious practice that affords little good and much potential evil. The only way that the body can derive water from infused salt solution is by renal excretion of sodium in higher concentration than it is administered. The kidney in the postoperative period may have lessened ability to concentrate sodium salts making available water even more difficult to provide.[9] The administration of sodium salts in the postoperative period should be limited to: (1) The replacement of extrarenal loss, the type of fluid administered depending upon the approximate constituents of the fluid lost and (2) replacement of extracellular fluid immobilized by ileus, mechanical intestinal obstruction, peritonitis, infection, tissue trauma and other conditions in which significant amounts of extracellular fluid may be sequestered and therefore are physiologically incapable of supporting circulation and other essential functions of the extracellular fluid.

The withholding of sodium salts when they are needed can produce serious disturbances in the relation of the concentration of the serum electrolyte to plasma water and protein particularly if infusion of glucose and water continues. As the sodium salt concentration of the serum falls there is decreased ability of the extracellular fluid to hold water and gradually water moves into the cells with resultant intracellular edema. Not only does this interfere with convalescence but may importantly contribute to an unhappy demise of the patient.

In recent years much has been written about the need of potassium salts in postoperative surgical patients. The postoperative diuresis of potassium is a natural phenomenon of the body's reaction to stress and attempts to replace this loss are not only futile but may be dangerous because of variables observed postoperatively in the concentration of the serum potassium. Large volumes of supportive intravenous fluids, however, increase urinary loss of potassium. During the first 3-4 days postoperative this, as a rule, has little significance. Administration of potassium salts in the early postoperative period should, for the most part, be avoided because of possible low urine outputs and actual elevation of serum potassium. If parenteral fluids are necessary after the 4-5 postoperative day they should be implemented by potassium salts.

Plasma and Blood

These is little to be obtained from plasma in the postoperative period that cannot be done better by whole blood. Plasma as a source of amino acids for nutritional purposes is inefficient and can be very expensive. Homologous serum hepatitis is still a too common complication of plasma transfusions. Whole blood is also an impractical and expensive method of providing protein for nutrition. Both plasma and whole blood do provide protein to help cover the acute protein loss in the exudates of severe infections such as peritonitis and gas gangrene.

Whole blood has the important added advantage of implementing the circulating red blood cell mass and by this means correcting anemia produced by immobilization of red blood cells and depression of hematopoesis by starvation and infection as well as providing the most efficient method of supporting the circulation. Mild degrees of anemia do not necessarily need blood transfusions as they can be easily corrected by resumption of oral intakes and supplements of iron.

Carbohydrate, Amino Acid and Fat Solutions

Carbohydrate may be administered as glucose, fructose or a combination of both. About 100-150 grams of carbohydrate are necessary during a 24 hour period to prevent ketosis.[16] In addition to this effect the infused carbohydrate also exercises a protein sparing action by negating the necessity of the breakdown of body protein to provide energy calories. Carbohydrate administered at a rate of more than 200 grams per day does not prevent further endogenous protein breakdown.[17] In short term fluid therapy it makes little difference as regards the concentration of the carbohydrate provided that enough is given to prevent ketosis and exercise the maximum protein sparing effect.

Various amino acid solutions have been advocated as a method of administering protein intravenously. In the great majority of well nourished patients the administration of such amino acid solutions, during the first 2-3 postoperative days, has little benefit as they may be rapidly excreted in the urine or deaminized and burned for energy.[18] Beneficial effects can be obtained during long term fluid therapy by the use of amino acid solutions. It must be remembered that 30-35 non-protein calories per kilogram of body weight has to be given with a minimum of 150 gms of amino acids if nitrogen retention is to be attained.[18] Considerable caution must be used in the administration of amino acid solutions when there is serious hepatic damage, as they may not be well tolerated under these circumstances.

Intravenous fat solutions have been prepared but an entirely satisfactory solution has not been made commercially available.

Summary

1. Glucose and water solutions should be used to initiate intravenous fluids at the beginning of operation rather than sodium chloride solutions as this will prevent unnecessary sodium salt loading.

2. There is no substitute for whole blood during operation, particularly if the operation is of considerable duration and magnitude and the blood loss greater than 500 cc.

3. The dangers of over-transfusion during operation have been described.

4. Postoperatively it would be ideal to provide exactly what is needed. Practically this is impossible because of the numerous variables in post-operative care, the individual handling of supportive fluids and defects in our knowledge.

5. Careful correlation of postoperative physical findings and laboratory examinations is necessary if maximum safety of parenteral fluid administration is to be obtained.

6. Administration of sodium salt solutions as a source of water should be avoided. Volume for volume replacement of extrarenal losses of fluid with sodium salt solutions of approximate constituents must be done if electrolyte depletion is to be avoided.

7. Amino acid solutions are of little benefit in the days immediately following operation in well nourished patients. They may be of considerable help in long term fluid therapy. In the presence of hepatic damage caution should be exercised during their administration.

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Anesthesia and Cesarean Section

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Cesarean section is an operation which calls for the closest teamwork between the anesthetist and the obstetrician. Each must be thoroughly acquainted with the problems facing the other in the management of seriously complicated obstetrical cases needing delivery by this method. Since the legal and moral responsibility for the welfare of both mother and infant belong primarily to the operator, it is quite natural that he should take the deepest interest in all of the problems, which are presented to the anesthetist. Ideally, each case should be reviewed together by the obstetrician and anesthetists, complications should be pointed out and idiosyncrasies against any of the commonly used anesthetic agents if known should be discussed.

The presence of cardiac disease, tuberculosis, diabetes, pneumonia and any number of less common diseases should be stated and their potential effect on the condition of both mother and baby carefully evaluated. In some cases premature delivery may indicate a special form of anesthesia and special attention to the newborn baby while in the operating room and also after removal to the nursery.

One of the first considerations when a cesarean section is to be performed is the type of anesthetic to be used. Ideally, the anesthetist and the obstetrician should both have an opportunity to express themselves regarding preoperative medication of the patient, the time of administration before the operation begins and the depth to which it should be carried.

If we consider first the advantages and disadvantages of the various anesthetic agents in uncomplicated cesarean section cases we can use this as a point of departure in discussing the operation when considering various complicated cases.

Choice of Anesthetic

In general, we believe on the basis of considerable experience that ether is the safest and probably the best anesthetic agent we have found. Nearly every anesthetist agrees generally on this point. However, it has the disadvantage of being irritating to the pulmonary mucous membrane and is therefore contraindicated when there is any serious form of lung pathology.

Chloroform is much less irritating to the lungs than ether, but has received a bad name because of use by inexperienced

Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 5, 1954.

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persons with little or no experience in giving anesthesia using the same technic of administration as would be used for ether anesthetization. It may best be used as a very light anesthetic which is all that is necessary in the great majority of cesarean section cases. The danger of late liver damage from chloroform has, I am sure, been greatly overemphasized.

Ethylene has in our experience been the best of the gas anesthetics. It has the explosion hazard which is objectionable but gives good relaxation if a small amount of ether is added.

Cyclopropane has the advantage especially to the baby of higher oxygen admixture but it has the same or greater explosion hazard as ethylene and I believe there is slightly more bleeding during its use.

Sodium pentothal may be used either alone or combined with general anesthesia. It has the disadvantage of personal idiosyncrasies in some cases and of the development of laryngospasm in others. Also the duration of anesthesia cannot safely be counted on for more than twenty to twenty-five minutes. Furthermore, it is readily diffused past the placental barrier and may seriously jeopardize the baby.

Of the various injection anesthetic methods local nerve block by pontocaine or novacaine yields the most satisfactory over-all results. It is true that delivery by cesarean section under local anesthesia is somewhat more uncomfortable, but it is much safer for the mother and baby than any other form of anesthesia. Many anesthetists as well as many surgeons seem to feel that there is nothing for the anesthetist to do during local anesthesia operations. This misconception is partly to blame for the unsatisfactory results obtained in some of these cases. Nothing can replace the comforting reassurance that a trained and sympathetic person is at hand and that the skillful addition of a small amount of general anesthesia to minimize certain painful episodes during the operation such as the extraction of the baby will be forthcoming.

The blood pressure must be carefully observed and early signs of shock detected and reported to the operator so that proper steps can be taken to combat the same before it becomes irreversible. Conversely, the surgeon should advise the anesthetist when to expect a reaction of the patient to some part of the operative procedure such as, pain, blood loss, or the discovery of unusual pathology which may prolong the operative procedure.

It is most important from the patient's standpoint that everyone in the operating room understands that she is fully conscious and understands all that is being said. Occasionally she misinterprets side remarks by the personnel which she thinks are directed towards herself. This is especially true of cesarean section operations because the preoperative morphine must be withheld on account of its serious depressing effect on the fetal respiratory center. The anesthetist can do much to offset this by distracting the patient's attention, warning the nurses and doctors, and in some cases we have felt that

cotton plugs in the ears to reduce auditory stimuli of all kinds are advisable.

Of the other forms of local anesthesia, caudal, spinal, and paravertebral, little need be said. They have practically no place in cesarean section. They are unnecessary because of the satisfactory results obtained with other agents which may be used and they carry an inherent danger and discomfort in a sufficient number of cases to warrant their disapproval. In fact, they have largely been given up in many of the large centers because the insurance companies have refused to defend malpractice suits arising out of alleged injury and death following such forms of anesthesia in obstetrical cases. We have never used these methods in my clinic at the University of Illinois, or in my private cases, except in an occasional instance where it seemed wise to take the calculated risk, to avoid greater danger from other forms of anesthesia.

Management of Complicated Cases

When the toxemic patient does not respond to medical management the pregnancy must be terminated. In those patients who are not in labor or who, if in labor, are not progressing satisfactorily, cesarean section is the procedure of choice. If convulsions have not occurred or are not apparently imminent, local anesthesia is probably the safest and best. If in the convulsive stage of the disease, general anesthesia using cyclopropane is the choice. The reason for this is that the convulsions are controlled by the general anesthetic and this makes the operation more speedy and safe for both mother and baby. It should also be remembered that pulmonary edema is a common concomitant condition in many of these patients and their ability to combat the toxemia depends on the oxygenation of their blood which in turn transports oxygen into the fetal circulation. It is obvious that air and CO_2 entering an edematous lung, compressed from below by the diaphragm which is pushed upward by the full term fetus and laterally by pleuritic fluid of a hydrothorax, will have difficulty in diffusing into the maternal blood. Obviously too, such a patient is not an ideal risk for general anesthesia. A minimum of the latter superimposed upon a well directed local anesthesia gives the maximum security. When the heart tones are very rapid or slow and irregular, the baby should be removed with maximum speed and a minimum of general anesthesia of any type. Atropine gr. 1/150 and open drop ether are the best, in my opinion, because if the case is an emergency, and vomiting does occur, it is less dangerous than when the closed mask is used. Against this must be balanced the advantage of higher oxygen with cyclopropane which might significantly reduce fetal anoxemia.

Most cases of placenta previa needing cesarean section do not present a picture of emergency and therefore the anesthesia may be given as one would use it in ordinary pregnancy. An important point to remember is that

we should expect postpartum hemorrhage in these cases also. For this reason the patient should be lightly anesthetized and brought out of the anesthetic as rapidly as possible and at the same time meet the requirements of the physician for relaxation and control of vomiting for he may have to do a manual removal of the placenta and pack the lower uterine segment before closing the uterus and abdomen.

Threatened rupture of the uterus during labor associated usually with the formation of Bandl's ring calls for immediate and deep anesthesia to stop uterine contractions and prevent the actual rupture. The mother may be going into shock and may have to have a blood transfusion before and after operative intervention or spontaneous delivery.

Ablatio placenta requiring cesarean section may be a most dangerous condition. This is especially true of the complete variety in which case the baby is dead and the uterus is filled by blood clots and liquid blood, the so called Couvelaire uterus. These patients are often in shock from painful distension of the uterus and from intrauterine hemorrhage. They also have bleeding into the peritoneal cavity from tears in the outer layers of the uterine muscle which may amount to 300 to 500 cc.

The anesthetist must know that such a uterus cannot be made to contract and that therefore it must be removed after doing a cesarean section to remove the dead baby from the uterus. The nicest judgment is necessary to provide the necessary relaxation of the patient to permit hysterectomy and at the same time limit the depth of anesthesia. Here too a combination of local and general anesthetic may be very desirable.

The immediate postoperative care is very important. Oxygen is the primary need of the patient and this can be supplied to her tissues by overloading the hemoglobin she has left in her circulation and by replacing the red cells lost through the hemorrhage or by collecting, straining and returning the blood and serum found in the uterine and peritoneal cavities. Stimulants for the cardiac muscle, artificial respiration and adrenalin injections all may be of value, and the anesthetist is in a strategic position to observe the general condition of the patient and to report signs to the surgeon.

Not all premature detachments of the placenta are complete and give the above picture. Many are partial separations and the fate of the baby depends largely on how much detachment has occurred when the patient is first seen and what facilities are available in the form of compatible blood, operating room facilities, competent surgeon and surgical assistance, and lastly but by no means least the cooperation of a competent anesthetist. In some cases it may be a race between the surgical team getting the patient properly prepared and operated upon and the separation of the placenta to a degree incompatible with intrauterine fetal life. Cyclopropane and oxygen combined with local infiltration would seem to be the anesthetic of choice with oxygen only given during the time of preparation

for operation in those patients presenting unusually rapid and weak fetal heart tones.

In cardiac complications requiring local or general anesthetic for cesarean section or a combination of these forms of anesthesia, we advise most of these patients to enter the hospital a few days previous to operation if possible for bed rest and, if necessary, digitalization before anesthesia is administered. If decompensation is imminent the operation may be done with the patient in Fowler's position. A high incision may be used, half of which is above the umbilicus and the incision into the fundus of the uterus constituting a classical type of cesarean operation. Following the delivery of the baby the patient's head may be lowered somewhat since decompression of the abdomen makes a pulmonary ventilation much more effective.

Placenta previa poses a problem for the anesthetist in two ways. First we may have an exsanguinated patient and, because of continuous blood loss, may be forced to operate before blood can be obtained in sufficient quantity to restore the circulatory balance. Under these circumstances it should be remembered that plasma, glucose, Ringer's solution and acacia solutions are valuable substitutes for temporarily sustaining the blood pressure and stimulating the heart.

The maternal blood loss is reflected in the fetal circulation and considerable information regarding the condition of the latter may be derived from auscultation of the fetal heart sounds.

Rupture of the uterus is probably the most serious complication which the anesthetist and obstetrican have to face together. There are two types; one occurs before labor sets in or in the early stages of labor and is due to disruption of a previous cesarean or myomectomy scar. The hemorrhage from these ruptures is usually not too severe because the rupture takes place through scar tissue. The other type of uterine rupture involves the lower uterine segment in a previously intact uterus and results in tearing of the lower uterine segment with, in many cases, damage to the uterine artery or to its ascending or to the descending cervical branch. These patients are frequently in shock and the baby is dead and frequently is found loose in the abdominal cavity. Shock is apt to be profound and very severe anemia is a constant finding. No time must be lost. One pint of blood is started as soon as the type and Rh is demonstrated. The abdomen is opened, the baby removed and hysterectomy performed. Knowing the nature and duration of such an accident before operation we feel that moderate oxygen inhalations help to prevent the patient's condition from deteriorating rapidly.

The emergency nature of the general anesthetic has been pointed out by consideration of the subjects we have discussed. I wish, for the purpose of emphasis, to point out that of 45 maternal deaths last year in down state Illinois, five were due to anesthesia. Most of these had a history of emergency operation undertaken for one of these

serious complications. The patients vomited during the anesthesia, aspirated the vomitus and either died immediately or within a few days of a pneumonitis which developed as a result of the aspiration of the acid gastric contents. I believe, therefore, that when there is any doubt as to whether the stomach is empty or not, that the passage of a stomach tube and aspiration of the stomach contents is indicated before the operation is started. Another safeguard would be to omit the attachment of the fastening straps of the anesthetic mask so that if vomiting does occur the mask can be released immediately and the vomitus suctioned from the pharynx and trachea. Artificial respiration may have to be practiced following the clearing of the respiratory tract and provision for this contingency should be made prior to induction of anesthesia.

Conclusions

From a consideration of these facts it is obvious that when a cesarean section is to be done a dual responsibility is placed on the anesthetist and on the operating surgeon: each must evaluate the problems of his own special field in connection with the management of the case, and at the same time appreciate those of his colleague. Free consultation between the two must be the rule before, during and after operation. Unlike other surgical procedures in most of these cases the interest of the fetus must be given almost, if not quite as much consideration as that of the mother. Indeed, in some cases the indication for operation is almost entirely fetal and therefore, our attention should be concentrated on it when the mother's condition is good and is likely to remain good under the circumstances surrounding the case.

In order to understand the proper use of anesthesia in patients delivered by cesarean section it is of the greatest importance in a given institution that there be a close liaison between the departments of obstetrics and anesthesiology. The giving of anesthesia and analgesia to patients in labor in the birthroom helps to acquaint the anesthetist with the obstetrical problems and their solution and thus makes cooperation with the surgeon more simple.

Intravenous Agents in Anesthesia

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Ever since man has interested himself in pain relief, he has concerned himself not only with agents but also in methods. We have been interested in finding the safest anesthetic agent for the patient; one which gives the surgeon the best operative conditions and still is pleasant for induction and has no aftereffects. To date no one agent meets these criterion. Needless to say, the perfection of an anesthesia does not lie in a drug alone. A great deal lies in the skill and artistry with which it is administered.

As we meet people today who are coming to surgery, most all of them make two requests. First they want to be "asleep" and second they would like the anesthetic agent given intravenously. Many of them ask for "Pentothal" by name. It has been proposed that this, the present anesthetic age be called, "The Era of Intravenous Anesthesia."[1]

Intravenous anesthesia has made the anesthetic experience less terrifying and more comfortable to the patient. With intravenous anesthesia we have, I believe, added some increased risks for the patient. I would like to again focus your attention on them.

An axiom promulgated by one of my first teachers of pharmacology 20 years ago is as true now as it was then. He said intravenous anesthesia carries with it the danger that, "Once the drug is put into the vein, you can't get it back out. One must wait until it is either excreted or detoxified. People differ in the rate of detoxification of drugs." If we keep this in mind and inject small quantities of the drugs and observe the patient's reaction to the various drugs, we can make anesthesia safer for the patient.

Barbiturates are used as induction agents for inhalation anesthesia. This use is justified since with minor increased risk to the patient and with very little inconvenience to the anesthetist, a pleasant induction can be accomplished. The intravenous barbiturates were soon found to be

1. Searles, P. W., and Lenahan, R. M.: Intravenous anesthesia. New York State J. Med. **48**: 1699-1702, Aug. 1, 1948.

Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 5, 1954.

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useful for short surgical procedures, and the tranquil recovery phase that most patients experienced following their use in these cases made them even more popular. Although the soluble barbiturates were satisfactory enough for minor surgical procedures, when used as the sole anesthetic agent for major operations requiring deep anesthesia for more than half an hour or so, they were soon found to have serious disadvantages. For some years pharmacologists as well as anesthetists have stressed the point that the barbiturates, while they are excellent hypnotics, have little effect on pain sensibility unless given in what is virtually overdosage. It seems wrong to use a barbiturate in order to subdue reflexes arising from pain stimuli. When used alone in long operations, comparatively large doses of barbiturates may be needed, and a prolonged recovery period results.

Because of inadequate chemical methods of estimation, investigators have been handicapped in studies on distribution and fate of the barbiturates. The methods have now improved and we know that with median hypnotic doses of thiopental, equilibrium between blood and brain is attained one minute after injection.[2] An intravenous injection of 400 mgm. of thiopental results in an initial plasma concentration of 26 mgm. at the end of two minutes. During the first few minutes after injection plasma level falls rapidly. Then it reaches an equilibrium and from there on it falls more slowly. With slow intermittent injections lasting up to an hour, the plasma level falls slowly and only 10-15% of the drug is destroyed per hour. This suggests that the brief action of intravenous thiopental as a single dose is due to redistribution rather than detoxification of the agent. Conversely after repeated injections the drug is relatively long acting because of its rather slow rate of destruction.[3] The initial high uptake of thiopental by the brain accounts for the rapid induction of anesthesia. The rapid recovery after a single intravenous injection of the drug is intimately related to the fall in plasma level brought about by the rapid redistribution of thiopental in the body. Studies on distribution of this drug show that liver and kidney contain higher concentrations than occur in plasma, but two to three hours after injection the highest concentrations are found in body fat. Levels of thiopental five to ten times those present in plasma may be found in the fat at this time. It takes about one to two hours for equilibrium to take place because of the poor blood supply of fat. This redistribution of thiopental is important, since it means that fat constitutes a reservoir in which the drug is stored and from there it is gradually released to be detoxicated relatively slowly by oxidation. The fat acts like a buffering mechanism which influences slightly the intensity of anesthe-

2. Shideman, F. E., and Gould, T. C.: Studies on tissue distribution and rate of metabolism of thiopental. J. Pharmacol. & Exper. Therap. **103**: 361-362, Dec. 1951.

3. Brodie, B. B., and others: Fate of thiopental in man and method for its estimation in biological material. J. Pharmacol. & Exper. Therap. **98**: 85-96, Jan. 1950.

sia, and much more the duration of anesthesia with this intravenous agent. The more fat there is in the body the more efficient is the buffering action, and the briefer the effect of a single injection of thiopental. One investigator[4] mentions the importance of taking into account the variations in fat content of the body, which in normal men varies from 7.2 to 22.4% of body weight and in normal women 12.6 to 28.5% of body weight. He suggests that we attempt to correlate the percentage of body fat with duration and intensity of action of thiopental.

The newer technics developed have also shown that the rapid brief-acting barbiturates are almost completely destroyed in the body, only traces of the unchanged drug appearing in the urine. No one has been able to show that there is a breakdown of the barbiturate ring. Some investigators have suggested that the sulfur atom of the thiobarbiturate is replaced by oxygen. However, recent studies of this barbiturate metabolism using thiopental containing S^{35} have not supported this thesis. The breakdown of the molecule seems to be at the side chain and with thiopental the oxidation of the side chain converts it to a carboxylic acid derivative. This derivative has none of the central nervous depressant action of the parent thiopental.

The metabolism of thiopental is slow and with large doses or repeated small doses, the resulting plasma level equilibrium is above the anesthetic level and is maintained by the reservoir of fat. With improved animal preparations recent work[5] appears to substantiate early in vivo and in vitro evidence that the liver is the primary site of thiopental metabolism and other tissues are quantitatively unimportant. The postanesthetic somnolence is due to persistence in the plasma of pentothal itself which is coming out of the fat reservoir. This then is the reason why thiopental's unsupplemented use as an anesthetic should be restricted to selected surgical operations of brief duration where only small doses are needed. It is safer to maintain the relatively stable barbiturate at a low plasma level for basal anesthesia, and to obtain depth of anesthesia with other agents when long surgical procedures are contemplated.

Anesthetists began doing just this not long after thiopental was introduced. They began using it in combination with nitrous oxide and oxygen. The patient was quite satisfied with this anesthesia, but the surgeon often felt he was not getting optimum operating conditions. With the introduction of curare, another intravenous agent, we were better able to satisfy his demands. Then the pendulum swung away from the thiobarbiturate and many anesthesias consisted of thiopental for induction alone and maintenance of anesthesia with nitrous oxide — oxygen and relaxation brought about by one of the several curare preparations. The use of curare in anesthesia has made

4. Keele, C. A.: Distribution and fate of anesthetic drugs. Proc. Roy. Soc. Med. **45**: 245-248, May 1952.

5. Meyers, F. H., and Peoples, D.: The positive role of the liver in the rapid metabolism of thiopental. Anesthesiology **15**: 146-149, March 1954.

life easier for both surgeon and anesthetist, but the question that remains to be answered is whether it has made anesthesia "safer" for the patient. Certain as yet uncompleted studies seem to show that the addition of curare to our anesthetic technic increases the hazard and has raised the mortality rate of anesthesia. It is reported that this bears no relation to who administers the anesthetic, be it technician, resident or physician anesthetist.

During the past few years much has been learned about curare. We know that there can be no average or standard dose. Some people are quite sensitive to its effects as a muscle relaxant, while others appear resistant. When a relaxant is added to the anesthetic mixture, many anesthetists believe it decreases the amount of thiopental needed. Most studies show that it has an effect on all muscles, not necessarily sparing those of respiration. Because of this, attention of the anesthetist has been focused on the patient's respiratory functions. Meticulous attention is paid to the airway, and since the introduction of curare many more patients have had the trachea intubated. Assisted and controlled respiration is much more common. It has been suggested[6] that the structure of anesthesia consists of the triad narcosis, analgesia and relaxation perched on its base of apnea. Good evidence has been compiled to show that apnea with controlled respiration also helps reduce the amount of anesthetic agent needed. Poorly conducted controlled respiration may be deleterious to the circulation.

This focus of attention on respiration as it is altered by relaxants should not make us forget another important and adverse effect these drugs may have. As well as blocking activity at the neuromyal junction, and producing muscle relaxation, they have inherent in them the ability to block sympathetic ganglia with resultant deleterious effects on the circulation. Patients develop unexplained hypotension of a severe degree. Some patients have profound circulatory collapse with doses of curare that do not necessarily paralyze the respiratory muscles. Curare may on occasion also have a central nervous system depressant action. While not producing unconsciousness it may block the passage of impulses from centers important in maintenance of both respiration and circulation.

We are, perhaps fortunate in having antidotes which can overcome some of the effects of overdose with the relaxants. Now with an antidote supposedly up his sleeve, todays' anesthetist is more liberal with his doses of relaxants. But in some instances the antidote fails to outlast the relaxant and at times the anesthetist is surprised to see adverse effects in the patient following injections of neostigmine, atropine or nikethamide. The anesthetist is at a loss to explain why the patient suddenly develops an asthmatic attack, a marked tachycardia, an elevated temperature, has a convulsive seizure or just simple atelectasis. Intravenous agents affect people in various

6. Gray, T. C., and Reese, G. J.: The role of apnoea in anesthesia for major surgery. Brit. M. J. 2: 891-892, Oct. 25, 1952.

ways and should not be used empirically. These effects are, on occasion, overlooked as attention is focused on respiration, and they could perhaps be one of the major causes in the increased mortality rate attributed to curare.

The newest member of the "relaxant family", succinylcholine chloride, has been presented to us as almost ideal from the standpoint of absence of toxicity. The drug is of great aid to the anesthetist and I should not like to be without it. Some people have, however, remained apneic for many hours after an injection of succinylcholine which in the average patient produces no apnea.

When succinylcholine was rediscovered in 1949[7] and recommended as a muscle relaxant, its short action was ascribed to its enzymatic destruction by blood. In 1952[8] it was shown that in man, of the two enzymes, (true cholinesterase in the red cells and the pseudo-cholinesterase in the plasma) the pseudo-cholinesterase is responsible for the hydrolysis of succinylcholine. It has also been demonstrated that there is an inverse relationship between the duration of apnea and the pseudo-cholinesterase level in the serum. Patients having prolonged apnea following the use of succinylcholine proved to have low pseudo-cholinesterase levels in the serum. It has been suggested that unduly prolonged response to succinylcholine be treated with transfusion of whole blood. Fresh blood is preferable, but even after storage whole blood contains some 30 units of pseudo-cholinesterase per ml. of plasma. Soon there will be a commercial preparation called "cholase" available as an antidote against succinylcholine apnea. It is an ethanol fraction of concentrated human plasma and is a subfraction of Cohn's globulin fraction IV-6. This should be the specific antidote for succinylcholine apnea. There are several reports in the literature of patients who have had prolonged apnea from succinylcholine whose serum, when tested, revealed normal enzyme levels. These people were suffering from cancer and inanition. They had altered electrolyte values in the serum, primarily low serum potassium. It may be that in patients like these, muscle paralysis is difficult to reverse because of a loss of excitability.

No adverse effects on circulation after injection of succinylcholine in man are reported. Animal experiments have shown that this drug will also block transmission at the ganglion. It is possible then to have hypotension occur and the type of patient mentioned above who has altered electrolyte values may well respond in an abnormal fashion.

As anesthetists continued the use of thiopental in small doses for basal anesthesia, nitrous oxide and oxygen for analgesia and curare for relaxation, opiates began to be used by vein to potentiate the effects of nitrous oxide. Intravenous morphine sulfate has been used for many years, but adverse effects on both respiration and circulation kept it in bad repute. The search for a suitable

7. Bovet, D., and others: Rend. Ist. Superior di Sanita 12: 106, 1949.
8. Evans, F. T.; Gray, P. W. S., Lehmann, H., and Silk, E.: Sensitivity to succinylcholine in relation to serum cholinesterase. Lancet 1: 1229-1230, June 21, 1952.

adjuvant to nitrous oxide and oxygen anesthesia led to the introduction of meperidine (demerol) in 1947[9]. From a structural point of view meperidine is in some ways similar to morphine and in others to atropine. Like morphine it has a piperidine ring and this probably accounts for its analgesic effects. Like atropine it has both antihistamine and anticholinergic properties. Meperidine in small intravenous doses produces analgesia without undue effect on the respiration and blood pressure of unanesthetized persons. Enough evidence is available for us to say that it does depress respiratory function during nitrous oxide and thiopental anesthesia, if one injects more than 25 mgm. at one time. If meperidine is diluted, and only 10-15 mgm. is injected slowly and repeated at 20-25 minute intervals the depression is not marked.[10] When given intravenously the maximal cerebral depressant effects of the drug appear to be exerted in three to five minutes. Meperidine has been shown[11] to decrease the ability of normal subjects to tolerate circulatory stress. This is also more prone to be evident in the anesthetized person, and can be guarded against by slowly injecting small doses of the drug. After intravenous meperidine there will also be a less adequate response to conditions imposing a strain on the compensatory mechanisms of the circulatory system, such as shock or hemorrhage. If chronic or acute blood volume deficit is present one is more likely to see hypotension. Changing the position of the patient on the operating table will magnify the circulatory effects, and with patients in the sitting position, one is apt to see marked hypotension. The causes for this action of meperidine are probably vasodilation either by a direct local action of the drug, through liberation of histamine, or by central vasomotor depression and perhaps by diminished "respiratory pump" mechanism. Meperidine has a weak narcotic effect and allows, in contradistinction to thiopental, a rapid return to consciousness with delayed postoperative pain.

Pharmacologists have drawn attention to a common action of a group of substances used by anesthesiologists. This group includes procaine, quinidine, atropine, the antihistamines and meperidine. They all have some degree of local anesthetic action, ability to relax smooth muscle and to lengthen the refractory period of the heart, an analgesic and an antihistamine effect. Meperidine has been used intravenously to treat broncho-spasm in man. It has a spasmolytic effect on the biliary tract, distal colon and rectum and is used by some anesthetists in such operations as hemorrhoidectomy and posterior colporrhaphy with good effect. The drug has been shown to have a rather selective action on the laryngeal reflex.[12] Using intra-

9. Neff, W.: Mayer, E. C., and Thompson, R.: Nitrous oxide anesthesia without hypoxia. Brit. M. J. 1: 1400-1404, June 17, 1950.

10 Johnson, P. D.: Pethidine as an adjuvant to nitrous oxide and oxygen anaesthesia. Brit. M. J. 2: 705-707, Sept. 22, 1951.

11 Ruben, H., and Andreassen, A. K.: Pharmacological effects of pethidine on larynx seen during intubation. Brit. J. Anaesth. 23: 33-38, Jan. 1951.

12 Mushin, W. W., and Rendell-Baker, L.: Intravenous pethidine and flaxedil in anesthesia for thoracic operations. Brit. J. Anaesth. 22: 235-239, Oct. 1950.

venous meperidine alone it has been possible to intubate the trachea without the complications of laryngeal spasm and cough. The action of meperidine on the heart has also been noted[11] and absence of pulse irregularities during thoracic and cardiac surgery has increased its use during these procedures. As with most drugs one does see adverse effects when intravenous meperidine is used. They consist of varying degrees of hypotension and respiratory depression. Small doses, injected slowly, minimize these complications.

We have recently had come on the scene an antagonist to the circulatory and respiratory depressant effects of the opiates.[13] The antagonist is a drug known as N-allylnormorphine. It is a most interesting drug. Administered to a normal individual, it acts as a depressant to respiration and circulation; injected into man prior to the administration of a large dose of morphine, it inhibits the respiratory and circulatory depressant actions of the opiate; injected into a patient narcotized from large doses of morphine or the other opiate derivatives, it produces a strong respiratory stimulation and has a beneficial effect if the patient is hypotensive. Although it has these stimulatory effects, N-allylnormorphine has little if any action on the sedative effect of the opiates. The drug has no effect on depression of respiration or circulation caused by the barbiturates or any of the anesthetic agents.

N-allylnormorphine is structurally identical with morphine except for the replacement of a methyl group. Meperidine is structurally different from morphine when the formula is written on paper and the relationship between the two molecules is obscure, but when three-dimensional models are constructed there is a close similarity of pattern, so we can expect this drug to antagonize meperidine. The site of action of N-allylnormorphine has not been defined but it is probably central. It may act in a competitive fashion with the opiates for certain receptors on cells of the central nervous system. It is possible for the drug to either occupy the receptor and block the action of the opiate, or it may displace the opiate. This is probably a non-specific action because the sedative effect of the opiate is not blocked or completely displaced.

Another drug capable of the same effects against some of the opiates is levallorphan tartrate (3-hydroxy-n-allylmorphinan.) It bears a relationship to dromoran similar to that of N-allylnormorphine to morphine[14]. It is an effective antagonist against respiratory depression produced by dromoran, nisentil, morphine and demerol. Here then we have two more drugs which can be used intravenously to combat toxic effects of agents used for anesthesia. Some anesthetists have been using these drugs prophylactically in an attempt to eliminate the respiratory depressant and

13. Eckenhoff, J. E.; Elder, J. D., and King, B. D.: N-allyl-normorphine in treatment of morphine or demerol narcosis. Am. J. Med. Sciences **223**: 191-197, Feb. 1952.

14. Hamilton, W. K., and Cullen, S. C.: Effect of levallorphan tartrate upon opiate induced respiratory depression. Anesthesiology **14**: 550-554, Nov. 1953.

hypotensive effects that intravenous opiates possess.

It would be interesting to know what specific structural unit of N-allylnormorphine and levallorphan causes the antagonism. If such a unit is found the chemists might be able to synthesize drugs which could have the same effect of antagonizing the respiratory depressant effect of the barbiturates or cyclopropane.

Conclusion

The technic and agents I have mentioned certainly present an advance in anesthesia to the patient. Induction of anesthesia is easy. The plane of anesthesia is such that many anesthesiologists and surgeons believe that reflexes are active, and these reflexes are beneficial in aiding the patient to maintain his physiologic equilibrium. The patient is better able to utilize his defenses to the greatest advantage against surgical trauma. Emergence from anesthesia is for the most part uncomplicated. One is tempted to rationalize that the period of somnolence provided by thiopental and the analgesia provided by meperidine, as well as the immediate postoperative amnesia they both provide, benefits the patient immensely and aids in a postoperative recovery free of complications.

I have discussed, in a very cursory fashion, one of the recent methods of anesthesia in use today. The method is simple (if we don't make a pincushion of the patient in an attempt to find a vein), the drugs are all potentially lethal agents, and it remains for us to see whether the technic is safer for the patient. I am always tempted to compare our modern drugs and technics to the experience reported by Alice Magaw, one of your associates, in 1906. You recall she produced anesthesia with ether by the drop technic 14,000 times without a death. One of my English friends once made a statement which I feel is still quite pertinent. He said, "Etherized patients rarely die—over-curarized patients just fade away."

Modern drugs and new methods of administration pose special problems for the anesthetist. Careful choice of the anesthetic agent, as well as special care in its administration, is of increasing importance with the wider selectivity now available.

Anesthesia for Children

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In discussing pediatric anesthesia, it might be wise to limit our thoughts to a few of the problems which confront us as clinical anesthetists.

The preparation of the child for his visit to the operating room will be considered first. Secondly, we will examine some of the technical problems confronting us in the conduct of the anesthetic. The final phase of the discussion will concern some of the physiological disturbances associated with anesthesia which we are in a position to prevent or to combat, thus providing a safer journey through the operating room for the child.

The preoperative preparation of the child begins at his home. Understanding and intelligent explanations by the parents can do much to dispel the fear of the unknown that is associated with the first visit to the hospital. The attending physician, with his close relationship to the patient and family, can initiate this phase of the education. Misinformation, as provided by older children or friends, is to be avoided.

Next is the preoperative visit of the patient by the anesthetist. Many technics have been devised by people working with children in an attempt to establish a proper, receptive state of mind for the anesthetic induction. Because of busy schedules it is many times impossible to make these visits, or at least to spend time enough to make them worthwhile. It is not easy to establish rapport with these little people during a hasty few minutes, sometime prior to surgery. If provision is made for adequate premedication, this visit, in most instances becomes unnecessary.

The ideal preoperative state is that of the drug induced hypnosis in which the child is very drowsy but still cooperative. In addition, a state of amnesia may be added so that the child has no recollection whatsoever of his experiences. When the child arrives for induction in this condition, the work of the anesthetist is made much easier.

Beecher and Cohen[1] have demonstrated that it makes no difference whether we use a barbiturate or a narcotic drug for premedi-

Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 4, 1954.

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1. Cohen, E. N., and Beecher, H. K., Narcotics in Preanesthetic Medication; Controlled Study; Report to Council on Pharmacy and Chemistry. J.A.M.A. 147: 1664-1668 Dec. 22, 1951.

cation. They are equally effective. With children, I believe, the barbiturates are superior to drugs such as morphine or codeine because they cause far less respiratory depression with comparable states of psychic sedation. They are very effective in producing the state of drowsy cooperativeness, especially when used with scopolamine. Scopolamine is as effective as atropine in its drying effect on secretions, and in addition, it effects a marked psychic sedation on the child as well as a period of amnesia for events in the preinduction period. Many of these children awaken from the anesthetic, following surgery, asking when they are going to have their operation. This certainly leaves the child with memories far more pleasant than those associated with an induction marked by physical restraint and forced submission to mask or needle.

The amount of the drugs used for premedication is usually determined by body weight. Children under twenty pounds get no premedication. The dosage range starts with the twenty pound child who receives pentobarbital grains 1/2 and scopolamine grains 1/350. The dosages are gradually increased to a point where the large child, weighing over one hundred pounds, will receive pentobarbital grains 1 1/2 and scopolamine grains 1/150.

In place of the above schedule of premedication, it is sometimes advantageous to use a rectal barbiturate such as thiopental. This method of inducing hypnosis is of value in children who will be having repeated surgery, in children who are behavior problems, and in children who are to have minor surgery, the drug being used as a basal anesthetic and supplemented with an agent such as nitrous oxide. The one problem that is difficult to overcome in the use of this technic is that of timing the administration of the drug. The optimum effect of the drug is reached in about twenty-five minutes. When the operations performed on a certain morning do not adhere to the estimated schedule, the difficulties of administering these drugs can be seen. However, if time is no problem, this technic provides excellent preoperative sedation. If thiopental is to be used rectally, the pentobarbital is omitted but the scopolamine is given according to the previously suggested dosages.

Thiopental is administered rectally in a ten percent solution, twenty to thirty minutes before the time of desired maximum effect. A cleansing enema should be given several hours prior to administration of the drug. The dose employed when hypnosis is desired is one gram per 75 pounds body weight. When used as a basal anesthetic, the dosage employed is one gram per 50 pounds body weight.

Premedication may be markedly reduced or omitted entirely in children of poor general physical state or in children who have illnesses that may increase the hazards of the anesthetic. Certainly each child must be considered as an individual case and the premedication determined by his individual needs.

The technical problems confronting us in anesthesia for chil-

dren differ in many respects from those found in anesthesia for adults.

The use of the open mask technic for the administration of ether provides a relatively safe, effective means for anesthetizing young children. For the individual who works with children only occasionally, it is an ideal method. However, even with a technic as simple as this, there are dangers which are many times overlooked. It has been shown[2] that the oxygen content under the mask falls to alarmingly low levels during the ordinary course of anesthesia. If supplementary oxygen is not administered, there is danger of hypoxia developing in the child. The addition of 500 cc. or 1 liter flow of oxygen per minute under an open ether mask throughout the conduct of the anesthetic will remove this hazard.

In using the ether blower for the oral pharyngeal insufflation of air-ether mixtures, as is commonly employed for tonsillectomies or similar operations, caution must be exercised if oxygen is to be added to the anesthetic mixture. Many times when the insufflation technic is used, respiratory obstruction is present. It may be due to a tongue retractor or to some other instrument or it may be due to blood or secretions in the airway. The obstructed airway will usually manifest itself by such changes as cyanosis, labored respiration, or increased pulse rate. However, if oxygen is added to the anesthetic mixture, many of these warning signs will be masked. The obstructed airway remains but the gaseous exchange of respiration is still inadequate and carbon dioxide excess develops in the child.

When using an anesthetic machine with a closed or semi-closed technic, we must keep in mind that children are not merely small adults. Hazards such as excessive dead space, valvular resistance and ineffective absorbtion of carbon dioxide are of even more importance in anesthetic management in children than they are in that of adults.

A new born infant may have a tidal volume of only 30cc. A child of 3 years of age may have a tidal volume of 150cc and a 5 or 6 year old child a tidal volume of 250cc. If we are to employ a closed technic in anesthetizing these children, we must employ equipment that is designed to meet their specific needs. The dead space must be kept at a minimum in order that these small tidal volumes are not reduced to ineffective levels. The valves, if present, must open and close easily and effectively. Carbon dioxide absorbing material must be fresh and efficient.

If all of these requirements cannot be met, it would be safer to employ an open method, reserving the closed technics for the occasional period of the anesthetic where it is essential.

During the actual maintenance of anesthesia, there are a number of physiological disturbances which may occur and which we are able to prevent or correct.

2. Faulconer, A., and Latterell, K. E., Tensions of Oxygen and Ether Vapor During Use of the Semi-open, Air-Ether Method of Anesthesia. Anesthesiology 10: 247-259 May, 1949.

Hypoxia is by far the most dangerous of the complications of anesthesia. Many times it goes unrecognized and the consequences are terrible. Haldane[3] stated the problem well when he said, "Anoxia not only stops the machine, but wrecks the machinery."

The causes of hypoxia are many. We are in a position, at the head of the table, to watch for and prevent such things as respiratory obstruction, depressed respirations, low oxygen content of the anesthetic mixture, and the anemic and stagnant types of anoxia associated with blood loss and shock.

Everyone is probably aware of the dangers of respiratory obstruction, and yet, many times we hear the crowing or bubbling of a patient struggling for survival in an adjacent room. This sometimes goes on for long periods with no apparent attempt on the part of the anesthetist to correct the obstruction. We must remember that any noisy breathing is obstructed breathing. It is our duty to prevent it.

With depressed states, respirations must be assisted to insure an adequate exchange of gases in the patient's lungs. A bag and mask with a supply of oxygen should be available and ready for use at all times.

If the hypoxia is due to inadequate oxygen in the inspired atmosphere, the answer is obvious. Whether a mixture of gases is being delivered from an anesthetic machine or whether an open method is being used, the answer lies in increasing the amount of oxygen available to the patient.

We understand the dangers of hypoxia occurring in an individual who has obstructed or depressed respirations. We must remember that respiration is a two way process. Any interference with the inward flow of oxygen to the child will necessarily impede the outflow of carbon dioxide, the waste product of respiration.

Carbon dioxide accumulation has many deleterious effects on the body. Respiratory acidosis results in hyperpnea and increased respiratory effort. Carbon dioxide excess may bolster a failing circulation, masking the impending shock.[4;5] At the finish of the anesthetic, as the carbon dioxide excess is dissipated, the resulting precipitous fall in blood pressure can mean the difference between survival and death in a poor risk patient.

Excessive amounts of carbon dioxide may exert a direct depressant effect on the myocardium. This condition is probably at fault in many instances of unexplained sudden death in the operating room, so called "Cardiac Arrest." Carbon dioxide accumulation constitutes one of the major complications of anesthesia.

Shock occurring during a surgical procedure is not unusual. In most instances, this condition

3. Haldane, J. S., Respiration, New Haven: Yale University Press, 1922, p. 118.

4. Dripps, R. D., Immediate Decrease in Blood Pressure Seen At Conclusion of Cyclopropane Anesthesia: "Cyclopropane Shock." Anesthesiology **8**: 15-35 Jan., 1947.

5. Buckley, J. J., et al, Postanesthetic Hypotension Following Cyclopropane: Its relationship to Hypercapnia. Anesthesiology **14**: 226-237 May, 1953.

is easily corrected by transfusing the patient with whole blood.

The administration of blood during surgery may present technical difficulties if adequate preparation is not made in advance. Venipuncture is difficult in very small children. A canula or plastic tube of the proper size should be inserted into a vein prior to surgery. The time to replace blood loss is during the time that it is being lost. It should not be postponed, to be done as a ward procedure following surgery.

Great care must be exercised in determining the amount of blood to be replaced. Certainly, all the blood removed from the operative site by suction apparatus should be measured. This, plus a careful estimate of the blood on the sponges and drapes should serve as a basis for replacement. It is easy to understand that an insignificant amount of blood loss in a large child or adult would be an overwhelming loss to a ten or fifteen pound infant. There can be no hard and fast rules for blood replacement. Each transfusion must have justification for the amount given.

In recent years it has been shown that deep anesthesia interferes with the body's mechanisms of defense against shock.[6;7] The combination of surgical trauma and deep anesthesia may place the child in a precarious state of circulatory depression. The same degree of surgical trauma in a lightly anesthetized child will not produce anywhere near the degree of shock.

Bearing this in mind, we should strive for only that level of anesthesia that allows the surgeon to perform his work in a satisfactory manner. To carry the child deeper than this is not necessary. In many instances the anesthetic requirements vary from moment to moment in the same operative procedure. We should attempt to suit the anesthetic level to the depth needed at any particular time, allowing the level to come up during those times that deep anesthesia is not necessary.

The problem of heat retention in the child undergoing anesthesia and operation is a real one. In many places, the child is still thought to be in dire danger unless dressed in several shirts and gowns and covered with at least one warm blanket.

Elevation of body temperature under even ideal conditions is not unusual. On hot humid days when excessive drapes and clothing are permitted, the body temperature may rise to frightening levels. In some instances this hyperthermia may be a vicious, irreversible process in which the temperature continues to rise in the post-anesthetic period and the child expires.

Even with lesser degrees of hyperthermia we can expect deleterious effects on the child. With a rise in body temperature, there is a corresponding rise in the metabolic rate. This in turn increases the demand of the body for oxygen and also increases the output of carbon dioxide. Any

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6. Hershey, S. G., Zweifach, B. W., and Rovenstine, E. A., Effects of Depth of Anesthesia on Behavior of Peripheral Vascular Bed. Anesthesiology **14**: 245-254 May, 1953.
7. Bennet, H. S., Bassett, D. L., and Beecher, H. K., Influence of Anesthesia on Circulation Under Normal and Shock Conditions. J. Clin. Investigation **23**: 181-208 Mar., 1944.

defect in the respiratory mechanism that we have discussed previously will be aggravated by hyperthermia.

This condition may be combatted by placing the child on a water cooled mattress[8], packing him with ice packs and removing all of the excess clothing and drapes.

Summary

With proper premedication, the child's visit to the operating room can be made an experience devoid of unpleasant memories, and the work of the anesthetist can be greatly facilitated.

By acquainting ourselves with the technical requirements and hazards encountered in administering anesthesia to children, we can provide a safer journey through the operating room for these little people.

Although the technics differ, the equipment varies, and the patients are smaller, the fundamental considerations are the same as with any anesthetic administration. We must maintain a clear airway, adequate respiration, and sensible level of anesthesia, give plenty of oxygen and maintain a good circulation.

8. Bigler, J. A., and McQuiston, W. O., Body Temperature During Anesthesia in Infants and Children. J.A.M.A. **146**: 551-556 June 9, 1951.

New Attempts in the Prevention of Erythroblastosis Fetalis

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The purpose of this paper is to present our experiences with ACTH and Cortisone given to pregnant women as an attempt to prevent the development of erythroblastosis fetalis in their offspring. The last decade noted considerable progress in the treatment of the already established erythroblastosis fetalis in infants. Previous attempts to prevent the development of the isoimmunisation with the Rh factor remained futile. The theoretically very attractive haptene therapy, the ethylene disulfonate medication, anhydro progesterone therapy and the use of various vaccines were all unsuccessful in preventing the development of the above mentioned pathological condition.

The premise under which ACTH and Cortisone were given was that adrenocorticosteroids have been reported as having the ability to prevent antibody formation. Case reports have also appeared in the literature in which acquired hemolytic anemia was alleviated by these hormones. Isoimmunisation with one of the Rh factors is necessary for the development of erythroblastosis fetalis. During pregnancy a positive Rh_0 antibody titer or positive Coombs test are qualitative indicators of the Rh sensitization of an Rh negative mother to the red cells of an Rh positive fetus. Therefore it was hoped that ACTH and Cortisone, by preventing the formation of antibodies, would also prevent or alleviate the damage to the fetus from Rh sensitization.

Material

Sixteen patients received ACTH and/or Cortisone during their pregnancy. The criteria for their selection was a bad obstetrical history due to losses from erythroblastosis fetalis, previous pregnancies with babies affected with erythroblastosis fetalis or a rising Rh_0 antibody titer. Three of the sixteen patients presented no history of difficulties due to Rh isoimmunisation in their previous pregnancies, but had a rising Rh_0 antibody titer. Thirteen women had previously given birth to one or more infants mildly affected with erythroblastosis. Eleven had lost one or more infants affected with eryth-

Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 3, 1954.

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roblastosis fetalis which were stillborn or died shortly after delivery. Three had given birth to stillborn infants diagnosed as hydrops fetalis. According to their past obstetrical history, patients were divided in two groups: Group one comprises five patients with no, or only mildly affected infants in their previous pregnancies. Group two is composed of eleven patients, all of whom had lost one or more infants because of the severity of erythroblastosis fetalis.

Fifteen patients presented a rising Rh_0 antibody titer prior to the onset of hormone therapy. This information is shown on tables 1 and 2.

NUMBER	NAME	Rh_0 Antibody Titers in Albumin					Coomb Test		Hormone		
		PRE Rx	3 DAYS	HIGHEST	LOWEST	DELIVERY	PRE Rx	ON Rx	TYPE	DURATION	AMOUNT
1	G.O.	1:256	—	—	1:0	1:0			ACTH	3 days	300 mg
2	D.W.	1:32	1:8	1:64	1:0	1:0	+	—	ACTH	3½ mos.	2520 mg.
3.	M.M.	1:512	—	—	1:4	1:4	+	+	Cortisone	11 days	375 mg
4.	G.A.	1:256	1:512	1:512	1:0	1:0	+	—	ACTH	11 days	640 mg.
5.	D.M.	1:8	1:0	1:64	1:0	1:32	+	—	ACTH	18 days	1060 mg

TABLE 1

Methods

Prior to the initiation of hormone therapy, patients were admitted to the hospital in order to obtain base values of various antibodies and electrolyte determination. In order to determine the responsiveness of the adrenal cortex, a Thorn test was performed. But for the first six patients, the intravenous modification of the Thorn test was done since this modification proved to be more reliable in pregnancy. A complete ABO, Rh and MN genotype was made both on husband and wife. Base line values were obtained from serum Na, KCl, fasting blood sugar, blood pressure and body weight. Rh_0 antibody titers were tested in saline, beef albumin and AB serum. Indirect Coombs tests were performed. These tests were done at frequent intervals during the entire period of pregnancy. Each patient was carefully supervised in the prenatal clinic for the appearance of albuminuria, excessive weight gain, edema and hypertension.

Administration of the Hormones

Thirteen of the sixteen patients received ACTH, one received Cortisone and two received both Cortisone and ACTH. ACTH used in this study was either short lasting or of the long lasting type which was dissolved in a gel depot solution (Adactar 32, or ACTHARGEL) or in polyvinyl pirolidone. These drugs

were administered intramuscularly in divided doses, or once daily. The short acting water soluble preparation of ACTH was occasionally used intravenously. Cortisone was used either intramuscularly or orally. Generally the doses varied between 75 U to 100 U of ACTH or the same amounts in milligrams of Cortisone given three to seven times weekly. Many patients were started on a low dosage which was gradually increased to 100 mg. or units daily. Tables 1 and 2 show the length of time each patient was treated and also the amount of the hormones administered. The shortest period of hormonal administration was three days prior to delivery, during which time 300 units of ACTH were given. The longest series of injections was over a period of six months and ten days. The largest amount given was 8350 units of ACTH and 6560 mg. of Cortisone. While no other index of "Therapeutic Value" could be obtained prior to delivery, a depression or maintenance of a low antibody titer and reduced eosinophile count, were the yardsticks by which the hormone administration was regulated.

Results

The antibody titers were depressed in thirteen of the sixteen patients treated with ACTH and/or Cortisone. In three patients we were unable to decrease the titer to zero level despite the increased amount of the hormone used. We were able to decrease the titer in those three patients

NUMBER	NAME	Rh_0 Antibody Titers in Albumin.					Coomb Test		Hormone		
		PRE Rx	3 DAYS	HIGHEST	LOWEST	DELIVERY	PRE Rx	ON Rx	TYPE	DURATION	AMOUNT
1.	VS	1 8	1 0	—	1 0	—	+	+	ACTH	3 days	325 mg
2	LL	1:128	—	1 64	1 0	—	+	—	ACTH	2 mos	1000 mg
3.	MR	1 256	1 16	1:256	1 0	1 8	+	+	ACTH	6⅓ mos	6365 mg
4	NH	1 8	1 0	1 512	1 0	1 16	+	—	ACTH	3 mos	3130 mg
5.	LF	1 32	1 8	1 512	1 0	1 32	+	+	ACTH Cortisone	4 mos 3 days	6405 mg 300 mg
6	MP	1 0	1 0	1 64	1 0	1 32	+	—	ACTH	1 mo 5 days	1520 mg
7	SB	1 64	1 0	1 256	1 0	1 256	+	+	ACTH	2⅓ mos	1400 mg
8	BW	1 32	1 1024	1 1024	1 0	1 256	+	+	ACTH Cortisone	3 days 4½ mos	300 mg 6560 mg
9	ML	1 64	1 16	1 128	1 8	1 128	+	±	ACTH	3 mos 18 days	4480 mg
10	HJ	1 64	1 32	1 128	1 32	1 32	+	±	ACTH	3 mos 22 days	8350 mg
11	MS	1 64	1 8	1 32	1 0	1 64	+	—	ACTH	19 days	760 mg

TABLE 2

only occasionally and at that time we believe we had prevented the titer from rising further. Of interest is the factor observed in another three patients with an elevated titer, whose titer decreased for three dilutions while receiving ACTH only to return to the same high initial titer one day after the hormone administration was stopped.

The Coombs test was strongly positive in fifteen patients before the hormones were administered. During the hormonal therapy the test was altered to a negative or equivocal one in seven patients. We believe that the height of the titer is not correlated to the severity of the erythroblastotic process. This presumption proved true in the cases under discussion, since two patients with low titers gave birth to premature stillborn hydropic infants. The adrenocortical function as tested prior to the onset of therapy by the Thorn test proved to be satisfactory in all cases. The electrolyte studies done during this investigation did not show significant alterations. The blood sugars were elevated only in two patients to 128 mg.%, and this was transient. No toxemia, hypertension or unusual edema and weight gain were observed but for one patient (H.J.) who gained 32 pounds.

Eleven patients were delivered of live babies, and five had stillborn infants. Of the former, two were delivered prematurely after a spontaneous onset of labor. Of the stillborn infants, two demonstrated hydropic changes. All the stillbirths were premature at the 7, 7 1/2 and 8 months of gestation. Of the eleven viable babies, three were Rh negative at birth, one, when retyped routinely in three months, proved to be Rh positive. Excluding the two Rh negative infants, all others were affected with erythroblastosis fetalis ranging from very mild in three to moderately severe in six. Five of these infants were given exchange transfusions, one had a simple blood transfusion. No losses were encountered in the group of infants born alive. Five mothers who had previously lost their newborns due to erythroblastosis fetalis left the hospital with live infants.

Discussion

It becomes clearly evident that we have failed to prevent the development of erythroblastosis fetalis. Therefore, an answer is expected to several questions.

1. Were the decreased antibody titers a true picture of the diminished antibody formation or was it a test tube blocking reaction?
2. Did the height of the antibody titer correlate with the severity of the erythroblastosis fetalis?
3. Did we alleviate the erythroblastotic process?
4. Were the mothers harmed in any way by the hormone administration?
5. Were the infants harmed by the hormonal therapy?

The work of Hayes and Dougherty showed conclusively that with the amounts of ACTH they used in animals, antibody formation is inhibited.

It may be that the failure to prevent the development of the antibodies of erythroblastosis fe-

talis is due to the relatively smaller doses of ACTH used in this study as compared to Hayes' work in animals. But whether an inhibition of antibody formation occurred at all is elucidated by the fact that in the three previously mentioned patients, the titers returned to the exact "pretreatment" levels after cessation of hormone administration. This was in keeping with Cunningham's communication that they were able to confirm apparent decrease in the titers unless trypsinised cells were used in which the titers remained unchanged. These observations made us believe that the decrease in titer was more apparent than real, and was occurring in the test tube as an antigen antibody blocking. Whether this partly occurred in vivo can be only speculated. The apparent beneficial results in the five infants who were born alive mildly or moderately affected with erythroblastosis fetalis, and whose older siblings were stillborn can be explained by this theory. At best we can hope that ACTH and Cortisone, although unable to prevent the antibody formation, and the development of erythroblastosis fetalis, could maybe alleviate the severity of the hemolytic process.

At present there is no evidence that ACTH and Cortisone are harmful if administered during pregnancy either to mother or infant.

Summary

An attempt was made to prevent the appearance of erythroblastosis fetalis by administering ACTH and Cortisone to pregnant women.

Sixteen patients were studied. They had previous infants with erythroblastosis fetalis or stillbirth due to the same process but for three who had a rapidly rising titer as evidence of isoimmunisation.

There was an apparent decrease in the antibody titer in thirteen patients. The reasons for this apparent decrease were discussed. Five babies died in utero between the 7th and 8th months of gestation. All surviving infants had evidence of erythroblastosis fetalis which ranged from mild to moderately severe, requiring exchange transfusions in five cases. Five mothers who had previously lost babies from erythroblastosis fetalis returned from the hospital with their infants. No harmful effects from the hormone therapy have been noted, neither on the mothers nor on the surviving infants.

Solution to Difficult Problems by Use of Special Technics of Anesthesia

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Anesthesia has progressed tremendously from the time when the patient was given a premedication some 30 to 60 minutes prior to the time of the operation, which was followed by the "pouring of the ether." The introduction of new agents and the development of more efficient technic has added immeasurably to the qualities of the anesthesia in so far as the patient is concerned. However, with this increase and adaptability of anesthetic agents and technics to the newer and more involved surgical procedures, we have added to the already great responsibility of the anesthetist.

Respiration is, and has always been, recognized as one of the most dependable criteria of the anesthetic level. Therefore, if we are to depend upon this basic sign as our best guide, we must familiarize ourselves with all of the modifying factors which may come into play.

A factor of greatest importance is the emotional state of the patient. Many times we see the excitable patient come to the operating room in a state of anxiety. This patient undergoes a difficult induction and excitement period. He continues poorly throughout the remainder of anesthesia and returns to his room to awaken after experiencing another period of excitement and delirium. Most of this, if not all, can be avoided by the proper application of certain drugs that we have at our command.

To avoid the problem described above it is much better to prepare the patient for the experience to be undergone. First of all a visit to the patient in the hospital the night before the operation should be made and careful evaluation of the anesthesia problem to be encountered should be made. A little reassurance at this time, from the anesthetist, will go a long way toward allaying the fears and apprehension of the patient. Then, too, after determining the type of anesthetic procedure to be used during the operation, the proper pre-medication can be intelligently prescribed. This will require a little extra time but it will also result in improved patient care as well as a more successful anesthetic procedure. The age old practice of ordering morphine sulfate gr. 1/4 and atropine sulfate gr. 1/150

Read before the Annual Meeting of the Tri-State Assembly of Nurse Anesthetists, Chicago, May 3, 1954.
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routinely without seeing the patient previously should by all means be discontinued.

In general all anesthetists appreciate the fact that usually the easiest anesthetic situation is that in which the patient is supine and level, with the operating procedure being confined to superficial work not requiring profound relaxation. Factors which add to the anesthetist's difficulties in maintaining the patient are many and varied. Deep anesthesia takes the patient into a state of depression which interferes with vital functions. Usually the periods requiring profound relaxation are short; but when an extrinsic factor to further hinder the respiratory mechanism is added the critical point of compensation may be reached. Steep Trendelenberg position places a heavy weight upon the diaphragm that tends to stop the diaphragm from moving downward. The weight is the weight of the liver, the intestines, the spleen, plus one to six abdominal packs, plus whatever force is exerted upon the retractor. In addition the usual postural circumstances are reversed which in itself is a burden. The handicap is great and the work required on the part of the body to maintain respirations is increased markedly. Some of these factors are virtually impossible to control but the depth of the position can be. Ideally the depth of Trendelenberg position used should not exceed 15 degrees. Certainly some flexibility should be exercised in this respect. On the other hand there are those patients that have difficulty tolerating even 15 degrees. Striking examples are obese patients and those who have cardio-circulatory disease.

The reverse Trendelenberg or Fowler position, when in excess, presents certain problems. There is the tendency to pooling of blood in the lower extremities in proportion to the depth of anesthesia, and the consequent degree of relaxation. The return circulation to the heart is impaired so that circulation in general suffers. Respiratory efficiency is dependent on good circulation.

The prone position is poorly tolerated. This is especially true in the adult. To use the prone position without deleterious effects, the following aids may be used. A small firm pillow placed under the shoulder girdle anteriorly places the weight of the chest on the upper less mobile portion, leaving the lower ribs free to move with respiration. To facilitate respiratory movement of the chest further, a pillow may be placed under the body to support the pelvis at the iliac crests. This again leaves the lower chest freer in its movements and also prevents excess pressure being exerted upon the abdominal contents and diaphragm.

The lateral or kidney position is a very difficult position in all respects: It is hard to establish, to maintain, and to have satisfactory respiratory movements of the chest. The weight of the thoracic cage is being borne on the lower lateral aspect of the chest to further limit free excursion; and finally the head down and foot down break of the table are added which stretch the body lengthwise. The last and final insult in this position is the

raising of the so-called gallbladder elevator into the costoiliac interval. Fortunately for both the patient and the anesthetist, the use of the elevator is being discarded more and more in both kidney cases and in gallbladder surgery. Rapid changes in position may cause rapid and profound falls in blood pressure. Occasionally respiratory failure can follow rapid change of position with equal rapidity.

The position commonly used which interferes with respirations to the greatest degree is the lithotomy position. In the unnarcotized individual, the vital capacity is reduced 18% (maximum inspiration and expiration). To the anesthetized patient this normal capacity is further impeded and the expected exchange of gases is greatly reduced.

The Airway

To the anesthetist the maintenance of a patent airway is of prime importance. It is important that this free airway be maintained post-operatively as well as during the conduct of anesthesia. The complete airway includes the respiratory tract from the lips to the alveolar membrane.

The lips can be a source of obstruction especially in the edentulous patient. Interference with the passage of air takes place during inspiration and there is usually very little or no hindrance to the air flow during expiration. The nasal passages may be occluded because of a deflected septum, polypi, allergy causing swelling of the nasal mucosa or adenoids with resulting obstruction. This is especially important to the anesthetist who is unaware of the existing lesions and tries to bring the chin up with her hand or tries to apply a tight fitting mask which results in closure of the mouth and a struggling or anoxic patient. Respiratory obstruction may follow as a result of some anesthetic agents such as ethyl chloride. This occurs when the water vapor of exhaled air from the patient's nose or mouth freezes because of the low temperature produced by the vaporization of the agent. The frost collects on the open mask and may then cause an obstruction. The frost should always be removed.

Obstruction is most common and frequent in the oro-pharynx. The flaccidity of the muscles of the tongue during profound relaxation causes the tongue to fall back into the pharynx and thus obstruct the airway. An abscess in the floor of the mouth, retropharyngeally or in the neck may obstruct the airway passages depending on its size and extent of the swelling. Most frequently, however, the obstruction is due to a collateral edema involving the tissues of the aryepiglottic folds. Edema of these folds will often completely occlude the laryngeal aperture. Foreign bodies such as dentures may also cause oro-pharyngeal obstruction.

Respiratory obstruction may result because of increased salivation and secretion of mucous due to inadequate premedication of drying agents such as atropine and scopolamine. The glottis may be the source of obstruction because of small foreign bodies

which pass the base of the tongue but are too large to enter the larynx. The obstruction here is usually complete. There may not be sound but the tidal exchange will be absent and diaphragmatic activity will then be excessive.

The vocal cords have the important task of protecting the lower respiratory tract from foreign bodies or material. They are capable of forceful, partial or complete adduction. The forceful adduction of the vocal cords is known as laryngospasm; and this condition is not rare. Stimuli which may bring on this condition are many and varied. The most common are too high concentration of anesthetic mixtures, especially ether administered to the lightly induced patient. Foreign materials or bodies in the larynx will cause laryngospasm. The vocal cords can also be forced into adduction by stimulating the recurrent laryngeal nerves. Stripping of the rib periosteum as in thoracoplasties or dilating the rectum under light anesthesia, or manipulation around the celiac plexus may reflexly cause laryngospasm of varying degrees.

Certain drugs such as the barbiturates enhance the laryngeal reflex and increase the tendency to laryngeal spasm. The barbiturate most responsible is probably sodium pentothal. An attempt to intubate a patient under sodium pentothal may well cause a laryngospasm. Even the passage of a pharyngeal airway in patients under light pentothal anesthesia will sometimes create a spasm.

If and when this condition arises, it must be treated promptly. If the cause of the laryngeal spasm is due to too high a concentration of the anesthetic agent, the agent should either be diluted with oxygen or withdrawn altogether. If it is due to manipulation the procedure should be halted until the patient is in a deeper plan of anesthesia. If the cause is a reflex spasm from the use of a barbiturate the spasm should be broken and another agent should be substituted. As a rule, if the spasm is not too severe, positive pressure with 100% oxygen should be used. Once a little oxygen can be forced past the adducted cords the relief of the anoxia will cause relaxation and the patient may resume spontaneous respirations. Spasm of the cords can usually be broken by forceful manual compression of the breathing bag and tight fitting face mask. We must make sure, however, that the breathing bag has been completely emptied of its anesthetic mixtures and refilled with 100% oxygen. When the spasm has been partially broken it would be wise to introduce carefully an intratracheal tube.

Obstruction can occur in the patient with paralysis of both vocal cords. This patient does well enough while awake when he is able to overcome this form of obstruction to some extent by voluntary efforts and the use of the accessory muscles of respiration; but during deep anesthesia when voluntary control is removed the obstruction becomes evident. These patients should be intubated prior to surgery;

and the intratracheal tube should not be removed until the patient has become fully conscious and is well able to compensate by the voluntary efforts and the use of the accessory muscles of respiration.

Obstruction may occur during thyroid surgery causing tracheal collapse especially where there is an attempt made to remove a large thyroid gland which has been pressing upon the trachea for some time causing a weakness of the tracheal rings. This difficulty should be anticipated and an intratracheal tube inserted before the operation.

In the large bronchi, obstruction is usually due to foreign bodies or materials. One of the more common causes of obstruction at this level is an excessively long intratracheal tube which most frequently lodges in the right bronchus and thus occludes the left bronchus. The anatomical position and size of the right bronchus favors the entrance of a long intratracheal tube as well as foreign bodies. It is well to remember that accidental endobronchial intubation is more easily accomplished in children because of the shortness of the trachea. The shutting off of the left main bronchus will, in this case, eventually produce a collapse of the entire lung. Unilateral "lag," particularly on the left side of the chest suggests too long a tube extending into the right bronchus. The tube should be withdrawn until it lies just below the cords. It might be well to mention here other obstructions caused by an intratracheal tube, since the placing of an intratracheal tube often gives the anesthetist a false sense of security;

1. The patient may bite down on the tube because the anesthetic is too light. This can be avoided by the insertion of a mouth prop.
2. The tube may be soft and twist or kink on itself.
3. The slip joint connecting the tube to the inhaler may be undersized and thus cause some degree of obstruction with increased burden on the physical effort of the patient.
4. Secretions may accumulate and occlude the lumen of the tube. This can be prevented by periodic aspiration through the tube by means of a small catheter and suction.
5. The tube may become obstructed by packs placed in the oropharynx with such force as to compress the tube.

Whenever an intratracheal tube has been passed, the thorax should be easily inflated when hand pressure is applied to the breathing bag, and the respiratory effort of the patient should not be out of proportion to the tidal exchange.

Obstruction to the passage of gases in the lungs may occur in those patients with emphysema and pulmonary fibrosis. In these patients the induction of anesthesia may be delayed owing to obstruction of passage of a gas or vapor from alveoli to the blood. The cause of this impaired gaseous exchange is not too well understood. It is felt that thickening of the alveolar and capillary walls must be the basis for the poor

exchange of gases. Recovery may be delayed for the same reason. Induction in such patients should be done cautiously and without hurrying; because it is important to remember that to increase the concentration of the anesthetic agent is not without danger. The high concentration will eventually reach the general circulation and cause excessively deep anesthesia, from which recovery may be slow because of the obstruction to elimination of the gas or vapor.

Other intrinsic lesions of the respiratory tract producing respiratory obstruction are lesions such as carcinoma and bronchiectasis. Empyema, where the collection of pus is great, will produce breathing difficulties as will also a hemothorax or pleural effusion. Fluid of any kind in the pleural cavity creates respiratory embarrassment in these patients. Care should be taken to avoid excessive depression or excessive premedication. One of the most common conditions in the chest causing embarrassment of lung movement is that of paradoxical breathing. The modern anesthetist is confronted quite frequently with this condition in chest surgery. Open pneumothorax following penetrating wounds of the chest or occurring in the course of intrathoracic operations will cause the lung to collapse and bring on the effects of paradoxical respiration. This phenomenon or paradoxical respiration, is caused when air exhaled from the sound lung does not pass wholly into the trachea; but enters the affected or collapsed lung and causes it to expand partially during the act of exhalation. In other words, when one inhalation occurs, air is drawn back from the collapsed lung into the sound one. In consequence the patient's sound lung is ventilated largely by vitiated air which is shuttled from one lung to another. There actually exists a tidal excursion between the two lungs so that the volume of this swinging or shuttled air reduces by a corresponding amount the tidal volume passing to and from the outer atmosphere by way of the trachea. It can be readily seen how such paradoxical respiration will soon affect the oxygenation of the blood. The condition is treated by administering oxygen under intermittent positive pressure, preferably through an intratracheal tube.

Effects of Respiratory Obstruction

Obstruction of the airway calls for an increased demand on the muscles of respiration. This increased effort on the part of the patient is needed to move the mass of air past the obstruction. The additional effort required to move air past the block will eventually tire the patient and bring on a state of shock. Pulmonary edema also may develop when the negative pressure within the thorax is increased in an attempt to move air past an obstruction. This increased negative pressure predisposes to transudation of fluid into the alveoli.

The active expiratory efforts required to move air past an obstruction creates a positive intrapleural pressure to which the thoracic cage in man is wholly unfitted. The diaphragm which

is a muscle of inspiration cannot resist an increase in intrathoracic pressure and is, therefore, displaced downward. This displacement of the diaphragm can be prevented only by contraction of the muscles of the abdominal wall. Operating conditions in the abdomen are then unsatisfactory as the abdominal muscles now are tense and abdominal contents are heaving from excessive diaphragmatic action. This situation is often misinterpreted by the anesthetist who believes it to be a state of light anesthesia. Increased concentration of anesthetic gases or vapor under these circumstances may well prove fatal.

Carbon dioxide is an end product of tissue metabolism which is often spoken of as a waste gas and must be removed from the body. It is also, however, a physiological stimulant to the respiratory center so that a certain amount is needed in the blood stream for continued respirations. The anesthetist is more commonly faced by a raised concentration of carbon dioxide in the blood rather than by a lack of it. The elimination of this gas is just as important to the anesthetist as is the ingress of oxygen. Obstruction of the airway will not permit the removal of carbon dioxide. It is well known that carbon dioxide in low concentrations acts as a powerful respiratory stimulant. However, in high concentrations carbon dioxide is irrespirable. When the normal tension of carbon dioxide in the arterial blood is increased the patient first responds by a progressive hyperpnea and by a rise in the systolic blood pressure, but as the carbon dioxide is a powerful respiratory stimulant, it cannot be allowed to accumulate for long periods or in high concentrations in the blood without eventually fatiguing the respiratory centers. Carbon dioxide also plays an important role in the acid base balance which may be subjected to many disturbing influences. If and when the airway becomes obstructed causing a retention of carbon dioxide in the blood, a gaseous acidosis results. Many authorities agree that acidosis accompanies most general anesthesias. The severity of the acidosis depends, to a great extent, on the amount of carbon dioxide retention. We must always bear in mind that the subject of carbon dioxide and its elimination is second only in importance to that of oxygenation.

No provision is made by the body for the storage of oxygen. It is, therefore, imperative that the airway be patent at all times. Hypoxemia (decreased blood oxygen content) and hypoxia (decreased tissue oxygen content) are closely related and occur in sequence, hypoxia being a result of hypoxemia. Hypoxia may result from:

1. A decreased oxygen intake.
2. An abnormal slowing of the circulation.
3. Reduction of the oxygen carrying constituents of the blood.
4. Inability of the tissues to use the available oxygen.

The initial response to mild hypoxia is an increase in minute volume of respiration with little or no change in blood pressure. With increasing severity of oxy-

gen lack, the symptoms become more pronounced and represent an attempt on the part of the body to maintain adequate oxygenation of the tissues especially to the heart and brain, by facilitating the transport of oxygen by the blood. Adequate oxygenation is most important for the maintenance of the blood pressure; thus a failing respiration constitutes a distinct danger signal. When oxygen want is acute, respirations respond with an increase in rate and depth. The action of this oxygen want is to paralyze the respiratory center and this quickly leads to respiratory failure. Hypoxia, however, increases the sensitivity of the carotid body.

The reflex stimulation of the carotid body caused by lowered oxygen tension will produce an increase in rate and depth of respiration, increased cardiac output, and rise in blood pressure. Peripheral blood vessels are constricted with those of the brain in an attempt to, adequately, oxygenate the central nervous system. The rise in blood pressure results in an increase in blood supply to the heart muscle itself. Thus a mild hypoxic state stimulates the respiratory and vasomotor centers to increased action. Although it is true that the principal function of the carotid body is to maintain respiration in states of hypoxia, we should also know that prolonged hypoxia will, ultimately, depress the carotid body.

Hypoxia is also responsible in a large measure for the formation of pulmonary edema. Hypoxia increases the permeability of the pulmonary epithelium to fluids and consequently encourges edema formation. A vicious cycle is thus established, hypoxia causing edema and the edema in turn causing further hypoxia.

Mild prolonged hypoxia causes headache, nausea, and depression in the conscious subject. Hypoxia plays a great role in post-anesthetic nausea and vomiting. As a result of moderate to severe hypoxia, the nervous system may suffer extremely, and with even fatal results. All degrees of severity may be encountered. The recovery may be slow and a comatose condition may continue for days or weeks. The body temperature becomes unstable, and marked muscle spasm, convulsions, or paralysis may occur. Loss of memory, mental incapacity and delirium may develop. These symptoms are due to irreversible damage to the highly specialized pyramidal and Purkinje's cells in the cortex of the brain. Petechial or gross hemorrhages may occur in these areas. Reflexes are abolished if anoxia exists for a few seconds and coma ensues if the cerebral circulation is interrupted from 6-8 seconds. Irreparable brain damage occurs if anoxia continues 8-10 minutes and longer.

The most serious effects of anoxia are usually upon the brain and myocardium. The myocardial coordinating mechanism is extremely sensitive to lack of oxygen. It has been estimated that even under normal condition this organ required five times the amount of oxygen needed by the skeletal muscle.

We must remember that the effects mentioned above resulting from obstruction to the airway

apply not only to the anesthetized patient but apply as well to the patient suffering from drug depression and coma. The patients do not die directly from the drug or the disease producing the coma but indirectly because of the hypoxia and the retention of waste gases. The anesthetist, therefore, has a useful role in the treatment of coma. Her daily observations of the anesthetized patient and her familiarity with the complications seen in these patients make her an invaluable assistant to the physician in treating these patients.

Anesthesia with volatile or gaseous agents can be secured only when the agent can reach the blood stream in sufficient quantity and concentration to effect the desired depression. The anesthetic state cannot be maintained if it is impossible to preserve the blood level of the agent, and excessive blood concentrations cannot be avoided if the patient is unable to eliminate the agent because of obstruction to the airway. Patients have died because obstruction of the airway did not permit adequate elimination of high concentrations of anesthetic agents in the blood stream. Therefore, I would like to make the following simple, but pertinent suggestions:

1. Choose the agent with which you are most familiar unless there is some definite contraindication to its use. Remember the agent is only as safe as the individual using it.
2. Pay particular attention to the premedication, taking into consideration the action of the drugs that have been used in planning your approach to as well as the conduct of the anesthesia. A little forethought may save you a great deal of second guessing.
3. There are some logical explanations for all of the unusual phenomena occurring during anesthesia. Search for the cause and make adjustments without delay. Procrastination has no place in the anesthetist's daily schedule.
4. Remember that the major responsibility for the life of the patient during the surgical procedure is delegated to the anesthetist. May we always be sure that we are worthy of that all important responsibility.

SUMMARY

Induction of anesthesia is prolonged when there is obstruction to the ingress of the anesthetic agent. We have seen patients go through awe - inspiring excitement phases because we were unable to reach an adequate blood level. We have often tried to explain this prolonged excitement stage to those present on the basis that the patient is an old alcoholic, that he has been inadequately premedicated or that he is emotionally upset.

We must always remember that no single factor in an anesthetic procedure is as essential to a safe and satisfactory anesthesia, regardless of the technic used, as the early establishment and maintenance of a perfect airway. As one writer so aptly states, "Take care of the airway and the circulation will take care of itself."

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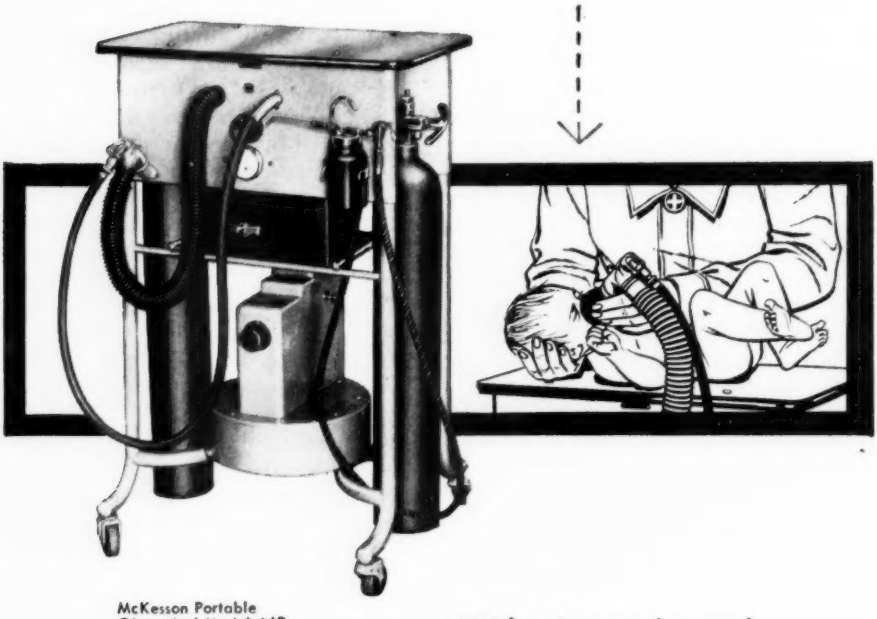

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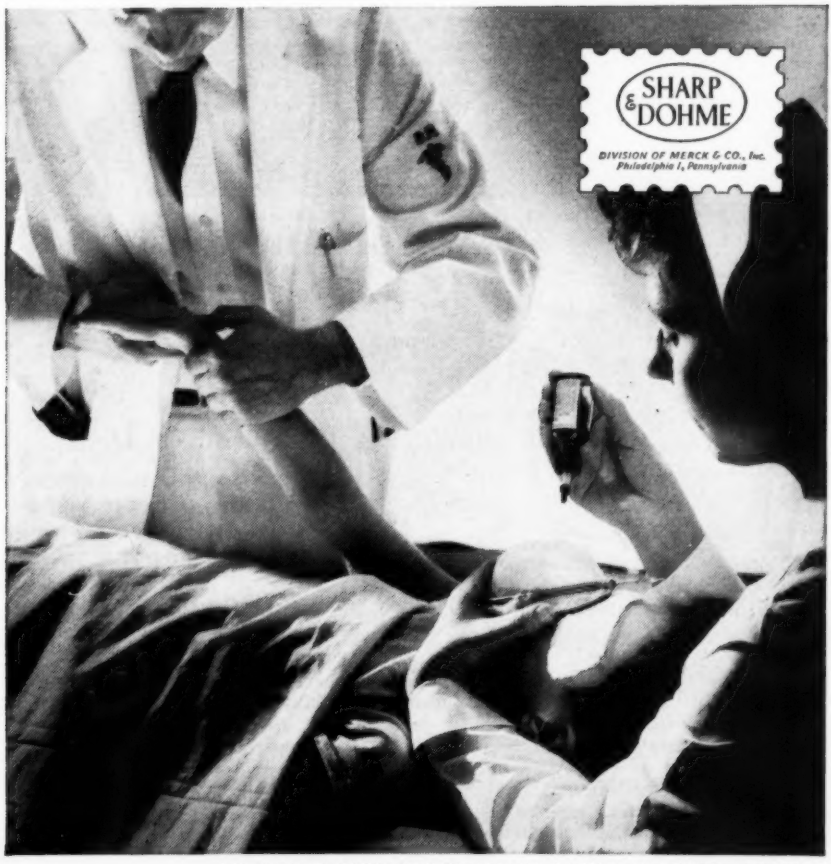

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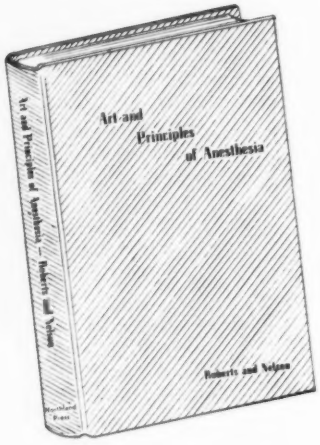

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Book Reviews

Anesthesiology by Forty American authors. Donald E. Hale, A.B., M.D., M.S., F.A.C.S., F.A.C.A., Head of Department of Anesthesiology, Cleveland Clinic, Cleveland, Ohio, Supervising Editor. Cloth. 722 pages, 149 illustrations. Philadelphia: F. A. Davis Company, 1954. $15.00

Forty authors have contributed to this book. The result is an excellent coverage of the subjects of interest to anesthetists. The supervising editor is to be commended for the high standard that he has apparently set for the material included in the volume. The method of having one or two authors present a specific phase of the specialty has in this instance resulted in a book that is far more interesting than many books written by one person. The wide range of subjects and the many authors make this a compilation of the best of current teaching and opinion in the field of anesthesiology. Some chapters have extensive bibliographies, others few or no references.

Psychodynamic Nursing. By Martha Montgomery Brown, R.N., B.S., M.A., Assistant Director and Assistant Professor of Nursing, Washington University School of Nursing, St. Louis; and Grace R. Fowler, R.N., B.S., M.A., Coordinator of Psychiatric Nursing and Assistant Professor of Nursing, Washington University School of Nursing, St. Louis. Cloth. 257 pages. Philadelphia and London: W. B. Saunders Co., 1954. $3.50.

In three parts of this book, the authors have presented Basic Concepts Utilized in Nursing, The Psychiatric Patient, His Treatment and His Care, and the Development and Trends in Psychiatry and Allied Fields. The material is presented for the use of student nurses and aims to help the student to "think reflectively and creatively in regard to the day to day situations which she meets."

Anoxia of the New-born Infant. A symposium organized by the The Council for International Organizations of Medical Sciences, established under the joint auspices of U.N.E.S.C.O. and W.H.O. Editors for the Council, J. F. Delasfresnaye, C.I.O.M.S., Paris, France and T. E. Oppe, St. Mary's Hospital, London, England. Cloth. 230 pages, Springfield, Ill.: Charles C Thomas, Publisher, 1954. $5.50.

This monograph is a collection of the papers and the discussions that were held in London, in October, 1951 on the subject of anoxia of the new-born infant. Seventeen authors from many countries participated in the symposium. Each paper and the discussion is independent of the others and the book does not pretend to cover the problem of anoxia of the new-born in its entirety. However, the presentation of their views by pathologists, obstetricians, physiologists and others who have studied the subject, will be invaluable to anesthetists who are vitally concerned with the problem.

Abstracts

Orr, R. B.: Anesthesia for encephalography and arteriography. S. Clin. North America **32**:811-816 (June) 1952.

"Since the technic of encephalography was first described by Dandy in 1919, many forms of anesthesia. . . . have been used. Since 1936, pentothal has been used almost exclusively at the Lahey Clinic for this procedure . . . . The use of routine premedication for these neurosurgical patients is to be condemned. . . . In most cases, atropine sulfate, 1/150 grain, is administered subcutaneously one hour before operation. . . . If signs of increased pressure are not present, and the patient is mentally alert, the addition of morphine sulfate, 1/8 to 1/6 grain, administered one hour before operation, appears to be of aid in the control of cough reflexes and in the reduction of the total amount of sodium pentothal given. The administration of nembutal in addition to morphine and atropine may be of advantage in the control of a particularly robust individual. . . .

"A series of 130 unselected cases has been reviewed. Of these, 23 patients underwent encephalography; 45 arteriography and 62, combinations of encephalography and arteriography. Pentothal sodium was used as the anesthetic agent in 103 patients, pentothal sodium plus decamethonium bromide (syncurine) in 24 patients and ether in 2 patients. Anesthesia was not used in one comatose patient. The average time consumed by this procedure was one hour; the longest, two hours and twenty-five minutes and the shortest, fifteen minutes. The average dose of pentothal sodium was 1.3 gm., the maximal dose, 3.8 gm., and the minimum, 0.4 gm. The average amount of decamethonium bromide used (in 24 cases) was 2.6 mg. In 13 cases, an endotracheal tube was introduced as part of the planned anesthetic procedure and in 3 cases an endotracheal tube was inserted as an emergency procedure. . . . Complications included laryngospasm in 4 cases, cyanosis in 3, vomiting in 2, tracheal deviation from hemorrhage in one, apnea in one and severe coughing in one. In one patient an acute respiratory obstruction developed a few hours after operation as a result of laryngeal edema for which a tracheotomy was necessary."

Auld, William: Simultaneous bilateral closed pneumothorax occurring during anaesthesia: Report of a case. Glasgow M. J. **33**:459-465 (Nov.) 1952.

"The production of open pneumothorax by accident or design is not infrequent during the course of surgical operations. . . . Case report: The patient was a male, aged 47 years. . . . The past

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history was relevant only to his peptic ulcer. . . . Sedation the previous night was with medinal gr. 7 1/2 and premedication was omnopon gr. 1/3 and scopolamine gr. 1/150. Anaesthesia was induced with pethidine 50 mg. and thiopentone 0.3 g. Tubarine 20 mg. was given, followed by nitrous oxide-oxygen mixture in the proportion of 85% N_2O to 15% O_2. Apnoea ensued and a cuffed endotracheal tube, size 9, was passed easily under direct vision . . . . Anaesthesia proceeded smoothly, and the abdomen was opened by an upper midline incision. Exploration revealed the presence of a large chronic duodenal ulcer but no other lesion was detected. The appendix was removed as a routine procedure. The time elapsing from the induction of anaesthesia until this point was 30 minutes. The patient's condition was excellent, and artificial ventilation was being carried out easily and with no muscular resistance. . . . While confirming the integrity of the endotracheal cuff by examining the pilot balloon, the patient's face was seen to become slightly pale. The pulse at the wrist was impalpable and the surgeon could not feel a heart beat through the diaphragm. . . .

"Cardiac puncture was attempted but the heart could not be found with certainty, no blood entering the syringe. It was now noticed that the left half of the diaphragm was bulging into the wound and the diagnosis of pneumothorax was made. A large bore needle was inserted into the thorax in the left mid axillary line in the 6th interspace and gas escaped under pressure. The needle was attached to rubber tubing, the distal end of which was placed under a water seal. The surgeon then felt feeble heart beats but they were not maintained. A second attempt at cardiac puncture was successful and three minims 1/1000 adrenalin were injected and the heart began to beat regularly, but at the rate of 36 beats to the minute. This cardiac action was maintained without further massage. . . . Despite the slow heart rate the patient's condition improved, and so he was allowed to remain undisturbed for half an hour in the hope that he would begin to breathe and the filling of the heart thereby improve and a normal rhythm follow. This did not occur, however. . . . During the previous 40 minutes no anaesthetic had been given and so anaesthesia was now very light. The tube was still in the trachea, and it was thought that with inadequate anaesthesia, the tube might be irritating the vagus, thus prolonging the heart block. It seemed unwise to remove the tube while the patient was still apnoeic and so an attempt was made to overcome any vagal inhibition by injecting atropine gr. 1/50 intravenously. The heart rate quickened immediately to 120 heart beats per minute. The condition of the patient now improved. . . . An X-ray of the chest while the patient was still on the operating table showed a large pneumothorax on the left side and a smaller one on the right. The case was therefore one of bilateral pneumothorax, occurring as a result of rupture of lung tissue

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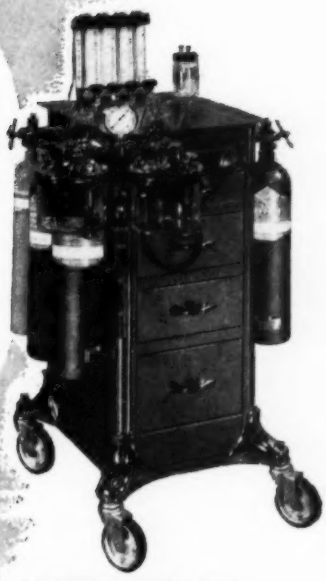

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. . . . Repeated X-rays over a period of four weeks showed gradual reabsorption of the gas and re-expansion of the lungs. . . . Three months later the patient returned and a partial gastrectomy was performed. . . .

"To provide maximum safety the anaesthetist should have a knowledge of the inflation pressure being used, the volume of gas being injected with each pulsation, and should have a safety device to blow off at not more than 18 to 20 mm. Hg. . . . The rarity of the condition suggests that anaesthetists are not using excessive pressures today."

Blatchley, Donald: Anaesthesia for tonsil dissection and adenoidectomy. M. Press **227**:395-398 (Apr. 23) 1952.

"A good anaesthetic can make a difficult operation much easier; a bad anaesthetic can turn the simplest case into a sanguinary shambles. In adults the anaesthetic problems are negligible once the anaesthetist has mastered the atraumatic intubation of the trachea; but in children the inexperienced administrator has many problems to face. . . . The majority of surgeons with whom the writer has worked prefer to operate without endotracheal anaesthesia, performing the adenoidectomy at the beginning of the operation with the pharyngeal musculature relaxed, and allowing the time spent in tonsillectomy for the adenoidal bleeding to cease. . . . The writer favours pre-operative sedation, with the recommendation of careful post-operative observation of the patient. . . . Atropine sulphate gr. 1/100 should be given by hypodermic injection to all patients 30 minutes before anaesthesia. For preference, the drug should be from a sealed sterile ampoule or freshly-made sterile solution . . . . For sedation, barbiturates hold the field of popularity. . . .

"It is very common for 'tonsil' children to have loose deciduous teeth, and these must be removed at the first opportunity after anaesthesia has been established before they become dislodged by the gag and possibly lost in the pharynx, oesophagus or bronchial tree. . . . Nitrous oxide has no unpleasant smell to the conscious, and if blown on to the patient's face from a distance produces a quiet and pleasant induction. . . . After a few breaths of pure nitrous oxide, oxygen is added up to 20 per cent. . . . Ether vapour is now gradually added to the mixture. A hand is kept on the mixture control and with each breath this is minutely advanced . . . . The patient has been inhaling ether vapour into the pharynx and tolerating it. The tongue-piece of the Boyle-Davis gag carries the anaesthetic delivery tube almost to its tip. This means that ether vapour is now insufflated directly into the larynx. Unless the larynveal reflexes have been obtunded, the result is laryngeal spasm. The experienced anaesthetist avoids this as follows: in the anaesthetic room he inserts the largest pharyngeal airway that will fit the patient. He then extends the patient's head by moving the pillow under the shoulders. This brings the tip of the airway forward and ether vapour impinges on the larynx in

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a similar way to that produced by the Boyle-Davis gag. This position is kept until the laryngeal reflexes have been lost and the ether vapour is tolerated. . . . The facepiece is kept in position until the last possible moment whilst the head towels are applied and the surgeon selects a suitable Boyle-Davis gag, to which a rubber delivery tube is attached. Then the facepiece is lifted, the airway quickly removed and the delivery tubing connected to the anaesthetic apparatus while the surgeon is inserting the gag. . . .

"For the first few breaths with the gag in position the strength of ether vapour should be reduced, then gradually increased to the maximum. As always in anaesthesia, the respirations are the chief guide. . . . At the conclusion of operation the Boyle-Davis gag is removed and a pharyngeal airway inserted. . . . Medical supervision must continue until vomiting has occurred or the recovery is so far progressed that the patient can vomit without the danger of inhaling it. When vomiting occurs the tendency to remove the airway must be resisted. . . . The one immediate post-operative complication which concerns the anaesthetist is that of intractable hemorrhage, and the surgeon's decision to search for and ligate the bleeding point. To do this he will need to insert the Boyle-Davis gag and will require anaesthesia. This is one of the most difficult and nerve-racking tasks that an anaesthetist can be called on to face, and fortunately it does not occur frequently. . . .

"Atropine gr. 1/100 should be given and no sedative drugs. The anaesthetist may be tempted to give sodium thiopentone into the blood drip to achieve smooth induction of anaesthesia, especially in the adult. This temptation must be resisted strongly. . . . Nitrous oxide and oxygen supplemented by the absolute minimal quantity of ether is the only anaesthetic to be considered. . . . The problem to be faced is to get the patient under sufficient control to tolerate the Boyle-Davis gag without inhaling any blood or clots. Before commencing, the patient is placed on the table and titled into Trendelenburg's position. The sucker is used to remove blood from the mouth and, if possible, from the pharynx. It may be possible to get the patient to tolerate a small urethral catheter passed through the nose into the nasopharynx, through which suction can be performed. Anaesthesia is induced and as soon as possible the Boyle-Davis gag is inserted and the pharynx thoroughly cleared of blood."

Legislation

Emanuel Hayt, LLB., Counsel, A.A.N.A.

Court Finds Evidence Does Not Sustain Claim Patient Was Dropped While Being Transferred To Wheel Chair—One evening, a woman stepped out of her trailer home, fell forward, landing at least on one outstretched hand, and suffered a severe fracture of her left wrist. She went to the hospital where her doctor placed the wrist in a cast. While being transferred from the cast table to a wheel chair by an orderly, she slipped or was dropped to the floor. Subsequently it was noted that she had recently sustained a dislocation and fracture of the right shoulder which was then treated by her physician.

The patient filed this action alleging that the shoulder injury occurred as a result of the negligence of the orderly. The court found as follows: the orderly was a servant and agent of the hospital; the patient was intoxicated, which was known to the hospital and orderly who failed to use reasonable care in handling her; as a result of their failure, she slipped from the arms of the orderly and fell to the floor; the injury to her right shoulder was not a direct and proximate result of such negligence. From these findings it may be inferred that the court found also that the shoulder injury occurred prior to her entrance into the hospital.

The patient now contended that the finding that she suffered no injuries as a result of the negligence of the hospital and orderly is without support in the evidence.

The trial court's findings are conclusive on appeal if there is substantial evidence to support them. The record showed there was such evidence.

(Morand v. Seaside Memorial Hospital, 3 CCH Neg. Cases (2d) 89—Calif.)

Immunity From Damages Denied To Charitable Institution Using Property For Commercial Purposes—The question here involved the tort liability of a private charitable organization. Defendant owned and operated for profit a four-storied building in downtown Kansas City, the total space of which was leased or rented to various tenants and in which defendant negligently maintained a common stairway for the use of tenants and their invitees.

Plaintiff, a business invitee of a third-floor tenant, who had sustained injury while using the common stairway, sued for $15,000 as damages. Defendant, a pro forma corporation organized for charitable purposes, occupied no part of the building but used all the profits from its operation for the maintenance and operation of a home for aged women.

"We have reached the conclusion" the court held, "that there never has been, and that there

should not now be established by court decision, a rule in this state which exempts or would exempt charitable organizations from tort liability where the activity out of which the alleged liability arose is or was wholly unconnected with, and not directly related to, the charitable enterprise for which the particular charity was organized and is operated; and that the fact, standing alone, that all the net profits from a charitable organization's commercial enterprise are used to carry on and accomplish the charitable activities and purposes of the organization, does not constitute or furnish the necessary direct connection or relation to call for the application of the immunity rule.

"Our holding goes no farther than to rule the specific facts of this case. What activity will or will not constitute the necessary connection with or direct relation to the charitable enterprise for which the particular charity was organized and is operated will depend upon the facts of each case. There will undoubtedly be some borderline cases. Each will have to be determined on its own particular facts."

(Blatt v. Nettleton Home for Aged Women, 3 CCH Neg. Cases (2d) 84—Mo.)

Court Affirms Judgment Against Employer For Injuries Caused By Pre-Employment Blood Test—Defendant was engaged in the business of baking and the manufacture of food products. Plaintiff applied for a position at defendant's factory; in accordance with the regulations of the Board of Health, plaintiff was compelled to submit to a blood test to determine the presence of any communicable disease. For this purpose defendant maintained a pre-employment room for examinations where it kept medical equipment and where it also employed nurses and a physician.

In attempting to get some blood from plaintiff, the doctor pierced plaintiff's left arm a number of times; these attempts were futile. Blood was later obtained from plaintiff's right arm.

As a result of the negligent insertion of the needle plaintiff suffered damage to the median and ulna nerves of the left arm resulting in a permanent claw-hand.

On appeal for a judgment for plaintiff for $30,000, defendant claimed that it was not derivatively liable for the medical neglect of the physician whom it employed. The court held that since the doctor was an employee the doctrine of respondeat superior was applicable. The doctor was performing work of a benefit to the employer, and defendant was liable for his acts. The relationship of physician and patient did not exist. Plaintiff was required to take the test from defendant's physician. She had no choice. In the absence of a physician-patient relationship the master is liable for the negligence of the physician who is acting for the benefit of his employer. The court thought that the amount of damages awarded was excessive by $15,000. The judgement for plaintiff was affirmed on condition that she stipulate to reduce the judgment to $15,000.

(Mrachek v. Sunshine Biscuit, Inc. 3 CCH Neg. Cases (2d) 80—N.Y.)

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WANTED: Nurse Anesthetists: Immediately. For 250 bed hospital. Permanent or summer relief. Paid vacations, Holidays, Social Security. Medical Anesthetist in charge. Contact: Sister Mary Concetta. St. Joseph's Mercy Hospital, Pontiac, Michigan.

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NURSE ANESTHETIST: General hospital, 700 beds; starting salary $4300 per annum, maximum $4800 per annum, $100 yearly increments; vacation and sick time; full maintenance provided; contact A. G. Chmelnik, M.D., Medical Director, The Harrison S. Martland Medical Center, 116 Fairmount Avenue, Newark, N. J.

Marion General Hospital, Marion, Indiana has opening for A.A.N.A. member, 100 bed hospital developing to 200 beds, good location, maintenance and laundry of uniforms, well qualified surgical staff, department of 4 anesthetists. Should make appointment by mid-September. Contact Administrator, Roy C. House.

NURSE ANESTHETIST—135 - bed hospital having 65 beds for medical and surgical patients and 70 beds for neurological patients. Complete maintenance available if desired. For details, write Mounds Park Hospital, 200 Earl Street, St. Paul 6, Minn.

NURSE ANESTHETIST: Well qualified nurse anesthetist wanted for 76 bed general hospital. Complete maintenance, salary open and liberal vacation. Give complete information as to education, experience and references in reply. Supt., Camden Hospital, Camden, S. C.

WANTED: Nurse Anesthetist—50 bed general hospital in Detroit - general surgical anesthesia, three day week with a rotating call, vacation and sick benefits, $500 per month starting salary. Box M-30, The Journal, A.A.N.A., 116 S. Michigan Avenue, Chicago 3, Ill.

NURSE ANESTHETISTS for 150 bed general hospital; four nurses, full time M.D., all agents and techniques; one month's vacation; two and one-half hours from Boston and New York. Write G. J. Carroll, M.D., Chief of Anesthesia Department, William W. Backus Hospital, Norwich, Conn.

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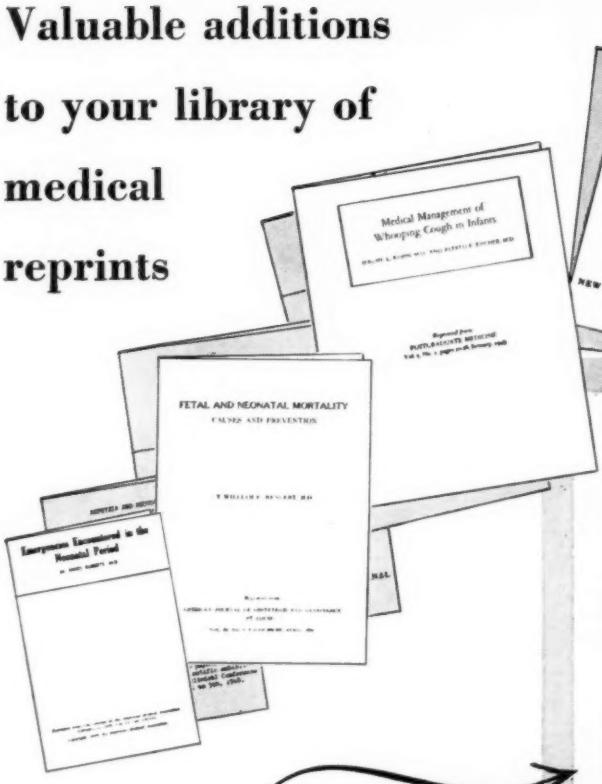
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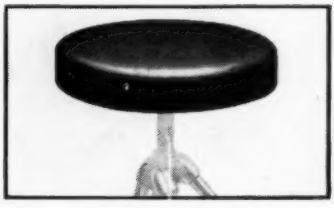

For further information please contact your OHIO branch or your nearest OHIO dealer . . . or write us for form 2073.

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